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Review of the National Ambient Air Quality Standards for Lead Exposure Analysis Methodology and Validation

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June 1989

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REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR LEAD: EXPOSURE ANALYSIS METHODOLOGY AND VALIDATION

OAQPS STAFF REPORT

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16. ABSTRACT

This report summarizes relevant information on multi-media lead exposure and presents the modeling methodologies that EPA staff believes should be considered for the review of the national ambient air quality standards for lead. Results of validating one of these methodologies are also presented.

The significant downward trends in gasoline lead emissions and lead concentrations in the diet are accounted for in these methodologies. Young children, pregnant women (as exposure surrogates for the fetus), and middle-aged men are identified as particularly susceptible to lead. Three different exposure methodologies, developed from a wide variety of data are described: the uptake/biokinetic and aggregate air lead models, both applicable to young children, and the disaggregate air lead model, which is applicable to both young children and adults. The uptake/biokinetic model allows explicit projections of future lead concentrations in different media and in turn can estimate the impacts of these changes on different age groups of children. It is this flexibility that makes the uptake/biokinetic model adaptable for a wide range of predictive exposure assessments and why it was the focus of the validation exercises described in this paper. Results of these exercises indicate good concordance between predicted and observed blood lead levels in children living near different lead point sources.

The uptake/biokinetic model can also be a useful tool in estimating exposures of children living with different lead hazards, such as heavily contaminated soils. Further refinements to the model are underway to allow applications to extremely high exposure scenarios.

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Air Quality Management Division
Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
Research Triangle Park, N.C. 27711

June 1989

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Helpful comments were also submitted by a number of independent scientists and by representatives of the U.S. Centers for Disease Control, the Lead Industries Association, the Canadian Department of Health and Welfare, the Canadian Ministry of the Environment, and the California Department of Health Services.

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TABLE OF CONTENTS

Page

List of Figures v

List of Tables v

Executive Summary vii

I. Introduction I-1

II. Lead Exposure: Multimedia Considerations II-1

- A. Airborne Lead II-3
- B. Lead in Soil II-5
- C. Lead in Dust II-9
- D. Lead in the Diet II-12
- E. Lead in Water II-13
- F. Lead in Paint II-14

III. Estimating Lead Exposures and Blood Lead Levels III-1

- A. Alternative Modeling Approaches III-1
- B. Calculating Blood Lead Distributions Around Predicted Means III-2

IV. Integrated Lead Uptake/Biokinetic Model IV-1

- A. Estimates of Lead Uptake IV-3
- B. Uptake and Blood Lead Concentrations IV-6

V. Statistical Relationships Between Blood Lead and Airborne Lead: Use in Disaggregate and Aggregate Models V-1

- A. Disaggregate Model V-3
- B. Aggregate Model V-14

VI. Validation of Integrated Lead Uptake/Biokinetic Exposure Model VI-1

- A. Validation Using East Helena Data VI-2
- B. Application of Uptake/Biokinetic Model to Other Data Sets VI-12
- C. Conclusions VI-17

TABLE OF CONTENTS (Continued)

Page

VII. Conclusions and Applications of Lead Exposure Modeling VII-1

Appendix A. Estimates of Lead Uptake A-1

Appendix B. Estimation of the Relationship Between Soil, Dust, and Ambient Lead B-1

Appendix C. Estimating 1990 Baseline Blood Lead Averages. C-1

Appendix D. CASAC Closure Report D-1

References

LIST OF FIGURES

Number	Title	Page
2-1	Principal Pathways of Human Exposure to Lead and Subsequent Physiological Distribution	II-2
2-2	Trends in Maximum Quarterly Lead Concentrations for Various Monitor Types	II-6
4-1	Summary of Relationships Between Daily Lead Uptake and Blood Lead for Different Age Groups	IV-17
6-1	Comparison of Distribution of Measured Blood Lead Levels in Children, 1-5 years of Age, Living Within 2.25 miles of E.Helena Smelter vs. Levels Predicted by Uptake/Biokinetic Model (Run B)	VI-8
6-2	Comparison of Distribution of Measured Blood Lead Levels in Children, 1-5 years of Age, Living Within 2.25 miles of E.Helena Smelter vs. Levels Predicted by Uptake/Biokinetic Model (Run C)	VI-9

LIST OF TABLES

Number	Title	Page
2-1	Typical Lead Concentrations in Various Exposure Media	II-2
2-2	Frequency Distributions of Maximum Quarterly Lead Concentrations by Type of Site	II-6
3-1	Comparison of Estimated GSDs Across Several Studies . . .	III-5
3-2	Illustrative Blood Lead Distributions Assuming Different Mean Blood Leads and Geometric Standard Deviations . . .	III-8
4-1	Estimated 1990 Average Lead Intake and Uptake in 2-Year Children Under Average Air Lead Levels.	IV-4

4-2 Predicted Equilibrated Blood Lead Levels Over Time Among Children with Constant Lead Uptakes IV-15

5-1 Disaggregate Model of Contributions from Various Media to 1990 Median Blood Lead Levels of U.S. Children (2 Years of Age): Background Levels and Incremental Contributions from Air. . . V-9

5-2 Blood Lead/Air Lead Slopes in Children for Different Ages and Exposures V-16

6-1 Comparison of Integrated Lead Uptake/Biokinetic Model Predictions to 1983 Measurements in East Helena VI-6

6-2 Children's Blood Lead Levels Measured in Omaha, 1971-1977 Vs. Integrated Uptake/Biokinetic Model Predictions. VI-14

6-3 Children's Blood Lead Levels Measured in Silver Valley, Idaho 1974-1975 Vs. Integrated Uptake/Biokinetic Model Predictions VI-16

A-1 Age-Specific Estimate of Total Dietary Lead Intake for 1990-1996 . . . . A-5

A-2 Total Dietary Lead Intake for 1978-1983 for Five Age Groups of Children A-5

A-3 Summary of Environmental Lead Measurements from Various Point Source Locations A-8

A-4 Long-Term Relationships Between Dust, Soil and Ambient Air Lead. A-13

A-5 Estimating Short-Term Responses in Equilibrium Soil and Dust Lead Levels from Changes in Ambient Air Lead A-13

B-1 Environmental Lead Concentrations in Omaha, Nebraska. . . . B-5

B-2 Linear Model Parameter Estimates for Air: Soil and Dust Lead Relationships. B-8

B-3 Average Dust and Soil Lead vs. Air Lead Relationships by Location B-8

C-1 Selected 1978 Blood Lead Levels from NHANES II C-2

C-2 Predicted Changes in Average Blood Lead Levels Associated with Gasoline Lead Trend. C-6

C-3 Trends in Dietary Lead Intake: Total Intake (and Atmospheric Contributions). C-8

C-4 Adjustments to 1978 Mean Blood Lead Levels for Dietary Lead Reductions C-11

C-5 Derivation of 1990 "Baseline" Average Blood Lead Levels . . C-14

C-6 Maternal:Child Blood Lead Concentrations and Ratios C-16

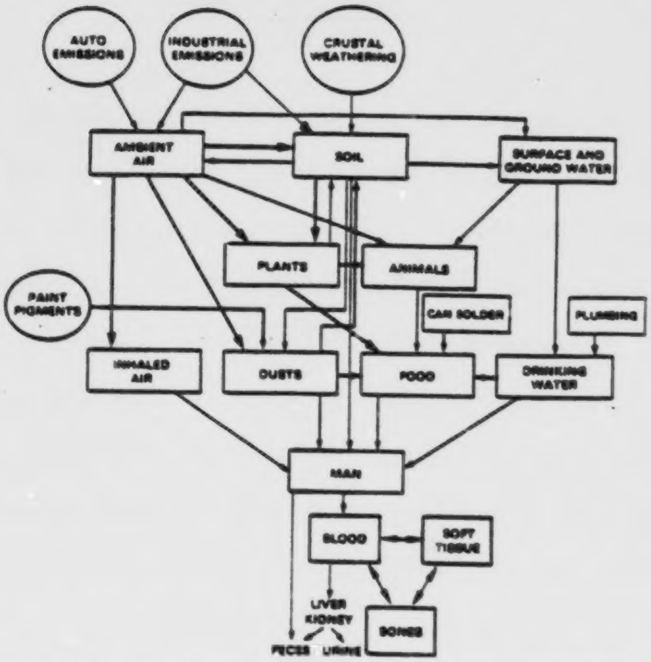
EXECUTIVE SUMMARY

For its review of the National Ambient Air Quality Standards (NAAQS) for lead, EPA is assessing health risks associated with lead exposure, particularly near stationary sources of atmospheric lead emissions (e.g., lead smelters), under alternative regulatory scenarios. A critical element in this process will be an exposure analysis whereby blood lead levels are estimated among populations exposed under alternative lead regulations in the future. This report summarizes relevant information on lead exposure and presents the modeling methodologies that the staff believes should be considered for the lead NAAQS exposure analysis. Results of validating one of these methodologies are also presented.

This report does not discuss health effects associated with lead, or the implications of different levels of lead exposure. That information is reviewed in the criteria document and will be integrated into a separate staff paper along with results of exposure analyses that employ the methodologies described here. The following discussion highlights the major points and conclusions discussed in this paper.

Multiple Pathways of Exposure

Human exposure to lead occurs through multiple pathways, and can be traced primarily to lead in paint pigments, solder in canned foods and plumbing, and atmospheric emissions from motor vehicles and stationary lead sources. Although airborne lead is a principal starting point of environmental contamination, oral intake of deposited atmospheric lead is often the primary identifiable factor predicting the blood lead of young children. The principal pathways of human exposure can be seen in Figure 1.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PLANTS
ANIMALS
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CAN SOLDER
PLUMBING
INHALED AIR
DUSTS
FOOD
DRINKING WATER
MAN
BLOOD
SOFT TISSUE
LIVER KIDNEY
BONES
FECES
URINE

Figure 1. PRINCIPAL PATHWAYS OF HUMAN EXPOSURE TO LEAD AND SUBSEQUENT PHYSIOLOGICAL DISTRIBUTION.

Downward Trends in Lead Exposure

Lead contamination of the human environment has been occurring for thousands of years since its many uses began to be discovered. Widespread, global dispersion of lead was greatly accelerated beginning in the 1920's with its introduction as an anti-knock compound in gasoline. Up until recently, approximately 90% of airborne lead in the U.S. was due to automotive emissions. Use of lead in gasoline has declined by about 90% since 1978; this trend will continue as compliance with the 1986 gasoline phasedown standard is completed and as the fleet of lead-burning cars shrinks. This trend has been paralleled by a major reduction in ambient air lead levels, which now average between 0.1 and 0.3 µg/m^3 in most U.S. cities without lead point sources.

The reduction in atmospheric lead, combined with the gradual phaseout of lead solder in canned food manufacturing and the ban of lead solder in plumbing has resulted in a significant decline in dietary lead intake. For example, it is estimated that from 1978 to 1985, average dietary lead intake in a 2-year child dropped from 52.9 micrograms (μg) per day to 10.4 μg/day. This trend is expected to continue.

These recent changes in nationwide exposure and expected future trends, as well as contributions from the other air and non-air sources of lead exposure (e.g., drinking water, contaminated soils) are important components of the methodologies described in this report. The air lead contributions include both direct inhalation of lead particles and indirect exposure through ingestion of lead in dust and in soil.

Lead-based paint, however, will continue to be the major source of high-dose lead exposure and symptomatic lead poisoning for children in the U.S. for years to come. High exposures to lead in older housing with deteriorating or even intact lead-painted surfaces (via chalking or weathering) will not be significantly influenced by any changes in atmospheric lead emissions. Because children excessively exposed to lead-based paint cannot be expected to be protected by any lead NAAQS, no matter how stringent, they will not be included in the exposure analyses described in this report. Available data are presented to demonstrate how such estimates could be generated. Comprehensive plans for safe and effective removal or containment of lead painted surfaces from old housing need to be developed and implemented.

Focus on Point Sources

The focus of the exposure modeling approaches described in this report, and the overall lead NAAQS review, is on stationary lead sources such as primary and secondary lead smelters and battery plants. Although such sources have contributed little to the overall pollution load across large, regional areas, fallout from these sources can be severe on a local scale. Ambient lead levels around such point sources vary depending on distance, topography, wind speed and direction, plant operating parameters, emissions controls, particle sizes and deposition rates, etc.

Data Limitations Preclude Quantitative Estimates for All Sensitive Sub-Groups

To assess the health risks associated with alternative air lead standards, it is necessary to estimate the blood lead (PbB) levels that would be distributed in the populations of concern under various air lead concentrations. Young children, pregnant women (as exposure surrogates for the fetus), and middle-aged men, are identified as particularly susceptible to lead-related risks. Quantitative estimates of blood lead distributions under alternative standards will be made only for young children and middle-aged men.

Fetal exposure to lead, in most cases, can be expected to be dominated by maternal bone lead stores from past exposures. Therefore, accurately predicting future changes in fetal blood lead levels would require estimates of maternal lead burden and information on how bone lead stores would be mobilized and transferred across the placenta. Although it is likely that there is extensive mobilization of lead, like calcium, during periods of physiological stress such as pregnancy, there are no biokinetic data to quantify this dynamic process. In the absence of such data, no attempt will be made to estimate fetal lead exposures associated with maternal PbB levels under alternative standards. Given the sensitivity of the fetus and neonate, however, potential risks associated with pre- and neonatal exposures will be of major emphasis in the overall lead NAAQS assessment.

Older women may also be at increased risk due to release of bone lead after menopause due to the processes of osteoporosis. A lack of biokinetic information on post-menopausal demineralization precludes quantitative estimation of blood lead changes under alternative lead NAAQS for these individuals as well.

Average Estimates Predicted by Exposure Models Will be Combined With Empirical Estimates of Variance to Calculate Blood Lead Distributions

This paper presents three approaches that can be used to estimate the impact of inhaled and ingested lead aerosols and compounds on PbB levels distributed in populations living near lead point sources. Because most of the data input to these models are generally average estimates, all three models first estimate PbB levels in terms of population mean levels. Given the significant behavioral and biological variability within populations, and the importance of highly exposed individuals within the population at risk, it is necessary to determine the entire blood lead distribution corresponding to a given mean PbB. This allows, for example, percentages of the population expected to exceed PbB levels of concern to be ascertained. Because PbB levels are typically distributed lognormally, it is possible to calculate percentiles of a blood lead distribution around a given mean PbB level by using the geometric standard deviation (GSD) of that population's blood lead distribution.

Several epidemiological studies have indicated that the log values of measured individual PbB levels in a uniformly exposed population are normally distributed with a variation, including analytical variation, ranging between 1.3 and 1.4. For purposes of predicting future exposures, a range of GSD values of 1.30-1.53 has been documented for children living near lead point sources, and the midpoint of 1.42 will be assumed as a reasonable best estimate. A GSD value of 1.37 derived from the NHANES II survey will be used for adults.

The three models are summarized below.

Uptake/Biokinetic Model: Applicable to Children from Birth

The first modeling approach presented is the uptake/biokinetic model which uses measured rates of absorption, or "uptake" of lead through different pathways (e.g., inhalation, ingestion) from experimental studies together with available mathematical ("biokinetic") models from lead balance studies to project either total body burden or the amount of lead in any of the presumed "physiological" kinetic compartments (e.g., blood, soft tissue, bone) at any time. The model is thereby capable of reflecting "non-equilibrium" lead exposures. It has been developed at this time only for young children.

The model is based on age-specific air, soil, dust, food, and water lead intakes, and on age-specific absorption factors. Blood lead and tissue lead distribution from absorbed lead is described by a linear pharmacokinetic model whose parameters change with age. The air lead concentrations around a point source can be estimated from historical data on emissions and site-specific information about meteorological dispersion parameters. To address situations when this information is not known, as in the case of assessing future NAAQS scenarios, a methodology was developed to estimate the associated soil and household dust lead concentrations using relationships derived from available data from a wide range of point source areas. This approach allows estimation of effects on soil and dust lead of historical changes or proposed changes in air lead levels. Dietary lead intake estimates are derived from historical and current data and future projections of the Multiple Source Food Model, originally developed in the 1986 criteria document, and updated to include more recent information.

Because the biokinetic model assumes linear rates of lead transfer between tissues, it is limited to predicting low-moderate level exposures (i.e., PbB < 25-30 µg/deciliter, or µg/dl). The model's mathematical assumptions and numerical parameters, however, combine plausible biological hypotheses, animal experimental data, and results of observational studies. Further, it allows explicit projections of future lead concentrations in various media and in turn can estimate the impacts of these different changes on different age groups of children. It is this flexibility that makes the integrated uptake/biokinetic model adaptable for a wide range of predictive exposure assessments, and why it was the focus of the validation exercises described in this paper.

Aggregate Air Lead Model: Applicable to Young Children

The second approach, referred to as an "aggregate" model, uses a directly applied mathematical relationship between air lead and blood lead derived from community epidemiological studies that includes both direct inhalation exposure and indirect exposures via secondary deposition processes. This approach requires an implicit assumption that dust and soil lead concentrations are approximately in equilibrium with air lead levels, and that the present air lead exposures reflect historical levels. This model also requires estimated contributions to children's blood from non-air sources of lead exposure, which are derived for current and future years based on such nationwide data as gasoline usage, dietary patterns, dietary lead concentrations, and blood lead surveys. Because aggregated air lead:blood lead relationships are not available for adults, and since the importance of indirect oral exposures to deposited atmospheric lead is relatively minor among adults, this approach is limited to young children.

This type of model is best used on a site specific basis due to the large number of confounders and covariates such as industrial exposure, age of housing, and social conditions. A summary of key studies useful in deriving aggregate blood lead/air lead slopes for children is included, along with a plausible range for these slopes (3-5 µg lead/dl blood per µg lead/cubic meter of air).

Disaggregate Air Lead Model: Applicable to Children and Adults

The third approach, referred to as a "disaggregate" model, is a hybrid of the first two that combines separate empirical relationships from multiple regression models between blood lead and lead intake from air, food, water, dust, and soil lead sources. This model was presented in the 1986 criteria document and is updated here to include more recent data. A number of experimental exposure and epidemiological studies, in which such data were available are discussed.

In this model, there is an implicit assumption that the soil and dust lead levels measured at some time reflect earlier exposures, so that the PbB levels estimated are essentially at equilibrium and not just reflecting some recent change or event.

The most plausible disaggregate relationship, or "slope" between inhaled air lead and children's blood lead, derived from analysis of 3 point source studies, is 1.97 µg/dl per µg/m^3. The average inhalation slope for adults derived from the most reliable experimental and epidemiology studies is 1.4 µg/dl per µg/m^3. A factor of 1.3 is applied to account for the resorption of lead from bone in adults and a slope of 1.8 µg/dl per µg/m^3 is derived. The slope values used in the "disaggregate" model are in general, consistent with those derived from the "uptake/ biokinetic" model.

Uptake/Biokinetic Model Validations Successful; Other Applications Should Proceed with Caution

Results of several validation exercises utilizing the uptake/biokinetic model comparing predicted and observed blood lead levels are presented in this paper. The most detailed analysis was performed with data gathered around the smelter in East Helena, Montana. Two types of validation efforts were undertaken with this data set: 1) in the first effort, the best historical data regarding such model parameters as measured air, yard soil, and household dust lead exposure estimates were used; 2) in the second validation effort, predicted levels of air (using dispersion modeling that accounted for fugitive emissions and background contributions), and soil and dust (using generalized relationships derived from empirical analyses of a wide range of point source data) were used to estimate PbB levels. The latter work was undertaken to determine how well the model behaved when actual measurements of necessary input data are not available. This was necessary so that the reliability of the model could be assessed for policy analysis purposes when less than full information is available to use with the model. Results of the different East Helena validations indicate good concordance between observed and predicted average PbB levels in children living near the smelter. The differences observed were not statistically significant.

Less extensive validation exercises on other published data are reported in this paper to test the uptake/biokinetic model at other U.S. locations and times including Omaha, Nebraska, from 1971 to 1977 and Silver Valley, Idaho, in 1974-1975. In these other validations, the model performed reasonably well in predicting average PbB levels below 25-35 µg/dl. As expected at higher exposure levels, the linear model underestimated actual exposures.

Given the many uncertainties in the input data and the biological variability that cannot be incorporated, the results of the validation exercises presented reveal that the lead uptake/biokinetic model performs quite well in predicting mean blood lead concentrations in children living near point sources of lead at exposure levels of current relevance. Using this methodology, along with the approach to estimate lognormal blood lead distributions using empirically-derived GSDs, exposure analyses of childhood populations living near stationary lead sources (i.e., "case studies") will be prepared. Blood lead distributions for middle-aged men in these areas will also be estimated using the disaggregate approach. Results of these analyses will be incorporated into the staff paper to better inform the Administrator on the impacts of alternative lead NAAQS.

The uptake/biokinetic model can also be a useful tool in estimating PbB levels in children living with different lead hazards, such as heavily contaminated soils from historical deposition near major urban roadways or closed smelters or mines. Ongoing regulatory efforts by different components of EPA to control concentrations of lead in air, water and soil have created a significant need to model blood lead concentrations that delineates specific routes of multi-media lead exposure. The "uptake/biokinetic" model adequately provides such specificity for young children. Further refinements are underway to include non-linearities in absorption rates and biokinetics. Until then, the model is limited to estimating relatively low to moderate exposures and should be used with caution in other applications.

REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR LEAD:

Exposure Analysis Methodology and Validation

I. INTRODUCTION

EPA is assessing health risks associated with lead exposure, especially near stationary sources of atmospheric lead emissions (e.g., lead smelters), under alternative regulatory scenarios for its review of the National Ambient Air Quality Standards (NAAQS) for lead. The scientific and technical information on lead has been reviewed in EPA's "Air Quality Criteria for Lead" (EPA, 1986). A "staff paper", in final preparation, will evaluate and interpret the most relevant of that information to help the Administrator in selecting the averaging times, forms, and levels for the primary and secondary standards.* A critical element in this process will be an exposure analysis whereby blood lead levels are estimated among populations exposed under alternative lead NAAQS in the future. This report summarizes relevant information on lead exposure and presents the modeling methodologies that the staff believes should be considered for the lead NAAQS exposure analysis. Results of validating one of these methodologies are also presented. Using methodologies described and validated in this report, exposure analyses of populations living near stationary lead sources (i.e., "case studies") are being prepared. Results of these analyses will be incorporated into the staff paper along with the overall health risk assessment. The staff will use results of the case studies to better inform the Administrator on the impacts of alternative lead NAAQS.

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*The current primary standard for lead (to protect public health) is 1.5 micrograms per cubic meter ($\mu g/m^3$), maximum arithmetic mean averaged over a calendar quarter. The secondary standard (to protect public welfare) is identical to the primary standard.

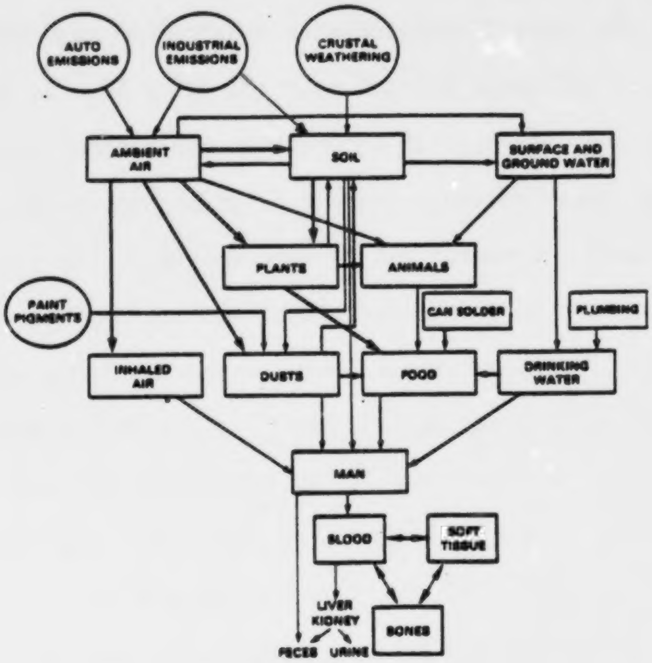
Normally, material of this type would be incorporated into the staff paper. Due to the complex nature of lead exposure analysis, we chose to place this material in a separate report to achieve a more thorough review and also to facilitate other applications of lead exposure analyses unrelated to the NAAQS review. This report should then be considered as a supplement to the OAQPS staff paper. It is intended for those readers familiar with the technical information contained in the criteria document (hereafter referred to as "CD"). This report does not discuss health effects associated with lead or the implications of different levels of lead exposure. That information is reviewed in the criteria document and will be integrated in the staff paper with results of the exposure analysis. This paper, as will be done with the staff paper, has been circulated for review by the Clean Air Science Advisory Committee (CASAC) and the public. A copy of the closure letter from the CASAC sub-committee that reviewed the August 1988 draft report is contained in Appendix D.

The approach used in this paper is to assess and integrate exposure-related information derived from the criteria document review that the staff believes should be considered in the review of the primary lead NAAQS. Section II presents relevant features of human exposure to atmospheric and non-atmospheric sources of lead through various pathways. Section III introduces different approaches to estimate the impact of alternative air lead levels on lead body burdens among different populations. Sections IV and V discuss the methodologies of these different approaches in detail. Section VI presents results of validation exercises using the uptake/biokinetic model. Section VII discusses some applications of the modeling approaches described in this report in addition to their role in the review of the lead NAAQS.

II. LEAD EXPOSURE: MULTIMEDIA CONSIDERATIONS

To assess the risks associated with alternative lead NAAQS, it is necessary to understand the influence exerted by atmospheric lead on the total lead exposure of the population(s) of concern through various exposure pathways. Relevant data on these pathways and the relationships between air lead and lead in other media are summarized in this section. The goal of the exposure assessments presented in this report is to address aggregate lead absorption from many pathways for populations living near stationary lead sources that will be affected by alternative lead NAAQS. Much of the methodology presented can also be applied to other populations with different exposure profiles. Exposure situations (other than operating stationary lead facilities) dominated by single, intense, and/or intermittent sources are not included for lack of adequate data, and/or because any lead NAAQS would have little, if any, protective effect for those situations. They include most significantly, high paint lead exposure (see Subsection II.F below) and occupational exposures. (Indirect exposures of children to occupational dusts tracked home by working parents are implicitly included in the modeling). Other miscellaneous intermittent sources not addressed here include various consumer products (e.g., leaching of lead-glazed pottery), and household activities or hobbies, such as stained glass construction and removal of lead paint soldering. See Section 7.3.2 of the CD for a more complete discussion of these kinds of exposure sources.

The sources and pathways of human lead exposure are diagrammed in Figure 2-1 and typical levels of lead in different media to which U.S. populations are exposed are presented in Table 2-1. Human exposure to lead can be traced primarily to lead in paint pigments, solder in canned foods and plumbing, as well as to atmospheric lead. Although airborne lead is a principal starting point of environmental contamination, oral intake of deposited atmospheric lead is often the primary identifiable factor predicting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DUSTS
FOOD
DRINKING WATER
MAN
BLOOD
SOFT TISSUE
LIVER KIDNEY
BONES
FECES
URINE

Figure 2-1. PRINCIPAL PATHWAYS OF HUMAN EXPOSURE TO LEAD AND SUBSEQUENT PHYSIOLOGICAL DISTRIBUTION. ADAPTED FROM CD, FIGURE 7-1.

TABLE 2-1. TYPICAL LEAD CONCENTRATIONS IN VARIOUS EXPOSURE MEDIA

Medium	Rural Area	Urban Area	Near Point Source(s)[a]	References
Ambient Air ($\mu g/m^3$)[b]	0.1	0.1 - 0.3	0.3 - 3.0	Faoro, 1988
Indoor Air ($\mu g/m^3$)	0.03 - 0.08	0.03 - 0.2	0.2 - 2.4	See Footnote c
Soil (ppm)	5 - 30	30 - 4500	150 - 15,000	CD, Table 7-11; Mielke et al.; 1983; LaBelle et al., 1987; See Table A-3
Street Dust (ppm)[d]	80 - 130 (90)	100 - 5,000 (1500)	(25,000)	Nriagu, 1978; CD, Table 7-26
House Dust (ppm)[d]	50 - 500 (300)	50 - 3,000 (1000)	100 - 20,000 (10,000)	U.S. EPA, 1977; Landrigan et al., 1975; Morse et al., 1979; Angle and McIntire, 1979
Typical Foods (ppm)	0.002 - 0.8	Same	Same	Flegel et al., 1988
Water ($\mu g/l$)	5 - 75 $\mu g/L$	Same	Same	Briskin, 1988
Paint[e]	<1 - >5 mg/cm^2	Same	Same	Billick and Gray, 1978

[a] Within 2-5 km of sources including primary and secondary lead smelters, battery plants.

[b] Represents quarterly averages monitored in 1986.

[c] Range of indoor/outdoor ratios used (0.3 - 0.8) from CD, Table 7-6 except near point sources where large particles predominate and infiltration into homes is low, ratio appears to be closer to 0.3 (Cohen and Cohen, 1980).

[d] Values in parentheses represent estimates provided in CD (Tables 7-18 and 7-20) as typical averages.

[e] Since there may be several layers of lead-based paint on a given surface, absolute concentration of lead is less useful than mg/cm^2. Surveys by HUD in Pittsburgh showed that more than 70% of pre-1940 dwelling units and 20% of post-1960 units had at least one surface with more than 1.5 mg/cm^2 lead paint (NAS, 1980).

the blood lead of young children. High correlations have been found between children's blood lead and the lead content of household dust, playground dust, mouthing behavior, lead on children's hands, and household cleanliness (Sayre et al., 1974; Baker et al., 1977; Charney et al., 1980; Roels et al., 1980, Yankel et al., 1977; Angle et al., 1979; Walter et al., 1980; Brunekreef et al., 1981; Quah et al., 1982; Bellinger et al., 1986; Bornschein et al., 1986; Schilling and Bain, 1988). Large scale isotopic studies provide strong evidence that air lead has a rapid and measurable effect on ingested lead, even in adult populations at low levels of air lead (Facchetti et al., 1985). It is necessary, therefore, to attempt to quantify how atmospheric emissions can influence human exposure, not only through direct inhalation of lead-containing particles, but through ingestion of lead deposited onto soil, dusts, vegetation, and other environmental surfaces.

Until recently, between 85-90% of airborne lead in the U.S. originated from gasoline combustion, with most of the remainder from stationary industrial processes such as primary and secondary lead smelting, battery plants, and combustion of oil, coal, waste oil and municipal waste. Because of the accelerated reduction of the lead content in gasoline and the continuing phaseout of older lead-burning vehicles, concern regarding lead emissions into the ambient air is shifting almost exclusively to relatively confined areas surrounding significant stationary sources of lead and this paper's focus will be on estimating exposures and risks in those locales.

A. Airborne Lead

1. Physical, Chemical and Spatial Characteristics

Ambient air lead levels result from current industrial emissions, lead-contaminated road and wind-blown dust, automotive exhaust, and solid waste combustion. The industrial contribution includes point, fugitive process, and materials handling emissions from lead mining, smelting, and recovery operations as well as various fabrication and manufacturing processes. Current contributions from roads and other fugitive dust sources vary with location and the influence of historically contaminated soil.

In general, about 50% of automotive lead emissions deposits within a few hundred meters of major roadways (Daines et al., 1970; Huntzicker et al., 1975; Ingalls and Garbe, 1982), while the remaining particles are small enough to remain airborne and can travel hundreds or thousands of kilometers. This long-distance transport likely accounts for the surface contamination of polar glaciers, oceans, and other remote locations around the globe (Settle and Patterson, 1982).

In contrast to automobile exhaust, atmospheric lead emissions from industrial plants that process lead and its products have contributed little to the overall pollution load across large, regional areas although fallout from these sources can be severe on a local scale. The trajectories of atmospheric emissions from industrial sources vary with wind speed and direction. High concentrations around lead smelters and other major emitters can be dominated by fugitive emissions, predominately made up of large [>7 micrometers (μm)] lead particles resulting from materials handling, furnace upsets, and furnace charging and tapping operations (Landrigan et al., 1975; Jennett et al., 1977; Battye et al., 1985). Stack and vent emissions can also produce significant and frequent short distance impacts, especially in complex terrain or around older facilities (Iaccarino, 1988). Beyond the immediate area (≈ 2 km) of lead stationary sources, process emissions from stacks, predominately as lead sulfates and oxides with a size range between 1 and 10 μm, become the major source of lead in soils and air (Dorn et al., 1976; Roels et al., 1980; Davidson and Osborn, 1984).

2. Ambient Concentrations

As indicated in the CD (Section 7.2.1) and EPA (1989), air lead levels in urban areas and near point sources have been markedly reduced since 1977 by the use of unleaded gasoline in new cars equipped with catalytic converters, the gradual phaseout of older lead-burning cars, the lead-in-gasoline phasedown program, and steady reductions in emissions from stationary sources in compliance with state implementation plans toward attainment of the lead and particulate matter NAAQS and partly because of decreased industrial production. Total atmospheric lead emissions dropped 94% between 1978 and 1987; gasoline lead use dropped by 90% over that period and industrial source emissions dropped by over one-half (EPA, 1989). Recent (1980-1986) air quality data for both point source-oriented sites (predominantly "SLAMS" sites), roadside sites ("NAMS", micro-scale) and other sites (middle-scale, neighborhood), are summarized in Table 2-2 (Battye, 1988). By 1986, the only quarterly average concentrations over 1.5 µg/m^3 were recorded at monitors near stationary sources. Twelve counties in the U.S. reported quarterly averages above 1.5 µg/m^3 in the 1986-1987 period; 13 more were on the margin of exceeding the NAAQS but either had inadequate numbers of samples or were slightly below the standard level.

Trends in maximum quarterly averages between 1980 and 1986 are illustrated in Figure 2-2. Even more dramatic declines are evident in data confined to roadside monitoring (see Battye, 1988 and EPA, 1989).

B. Lead in Soil

The natural occurrence of lead in the earth's crust averages 5-50 µg lead/g soil in various soils* (CD, p. 7-28), although soils near

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*One µg/g is equivalent to one part per million or 1 ppm.

TABLE 2-2. FREQUENCY DISTRIBUTION OF MAXIMUM QUARTERLY AIR LEAD CONCENTRATIONS ($\mu g/m^3$)[a]

Site type/time frame	Percentage of sites in concentration ranges (concentrations in $\mu g/m^3$) <0.5	0.5-1.0	1.0-1.5	1.5-2.0	>2.0	Mean	Number of site-years
1980 through 1986							
All monitors[b]	77.5	15.4	3.2	1.5	2.3	0.39	3,279
NAMS[c]	76.9	17.2	4.7	1.1	0.2	0.41	471
SLAMS	75.4	16.7	2.8	1.7	3.4	0.38	1,158
Microscale roadside[d]	24.1	37.9	25.9	10.3	1.7	0.74	58
Middle scale	45.7	47.1	7.1	0.0	0.0	0.46	70
Neighborhood scale	68.7	24.3	7.0	0.0	0.0	0.37	115
1986 only							
All monitors	93.0	2.5	1.2	1.6	1.8	0.17	514
NAMS	97.5	1.3	1.3	0.0	0.0	0.14	79
SLAMS	92.9	2.2	0.9	1.3	2.7	0.21	226
Microscale roadside	100.0	0.0	0.0	0.0	0.0	0.18	9
Middle scale	100.0	0.0	0.0	0.0	0.0	0.12	9
Neighborhood scale	94.4	0.0	0.0	5.6	0.0	0.14	18
1987 only							
All monitors	93.0	2.8	1.7	0.4	2.0	0.20	458
NAMS	97.4	0.0	1.3	0.0	1.3	0.20	78
SLAMS	93.1	2.3	1.7	0.0	2.9	0.20	175

[a] Data are from the SAROAD and AIRS systems and represent the number of site-years for which the maximum quarterly concentration fall within the designated ranges. To be included, a valid site-year must have at least three quarters of data with at least 6 observations per quarter.

[b] Includes sites previously classified into categories (e.g., "urban") that do not meet any of the current definitions.

[c] NAMS refers to the National Ambient Monitoring Station network, while SLAMS refers to the State and Local Ambient Monitoring Station network. Each of these two networks includes both roadside and point source monitors; however, in the case of lead, the NAMS network tends to focus on roadside sites, while the SLAMS network incorporates more point sources and other special purpose monitors.

[d] Microscale sites were within 5-15 meters from a major roadway. Middle scale sites had further setbacks and define concentrations up to several city blocks. Neighborhood scale sites define concentrations in more extended areas of uniform land use within cities (0.5-4 km^2). After 1986, data collection at these sites stopped.

Source: Battye (1988).

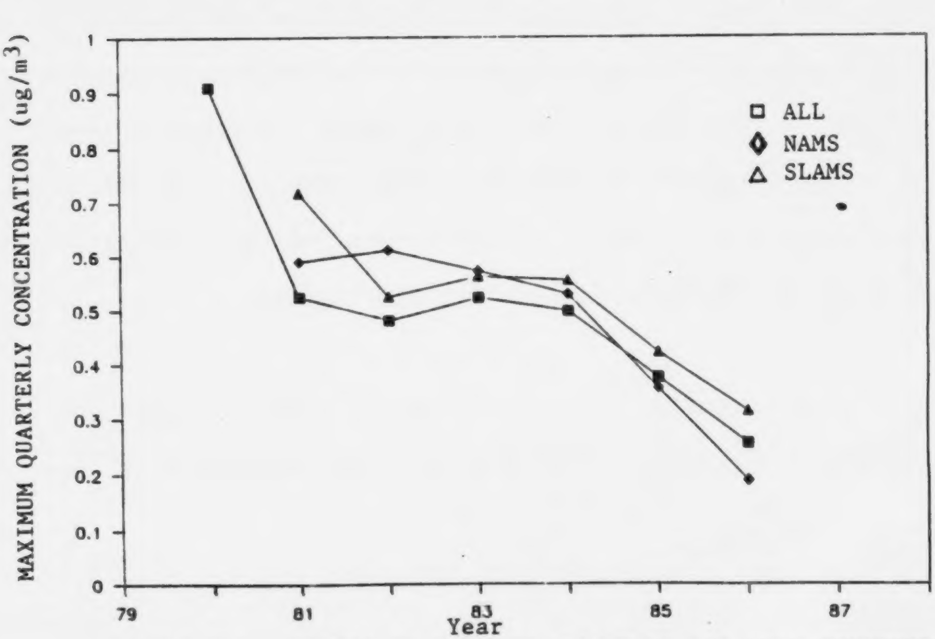

FIGURE 2-2. TRENDS IN MAXIMUM QUARTERLY AIR LEAD CONCENTRATIONS FOR VARIOUS MONITOR SITES, BAITYE (1988).

naturally occurring outcrops can reach several hundred ppm. Urban soils are contaminated by lead from a combination of automotive and point source deposition as well as from lead in paint on indoor and outdoor surfaces. Elevated soil lead concentrations (as high as 2000 ppm) have been found within 10 feet of wood frame houses painted with lead-based paint (Ter Haar and Aronow, 1974). Accumulations of lead in soils (and dusts) around brick or stone structures have also been found, and can be partially attributed to washoff lead collected on roofs, ledges, and exterior walls (Wheeler and Rolfe, 1979), and possibly to an aerodynamic effect of the structures on deposition of lead from nearby sources. Recent data from Baltimore (Mielke et al., 1983) and in and around Chicago and "downstate" Illinois (LaBelle et al., 1987) indicate that lead accumulates in soil away from painted structures and known industrial lead sources, in a pattern at least partially related to traffic activity and distance from nearby roads.

The upper layer* of roadside soils may contain deposited atmospheric lead in concentrations of 10 to 500 ppm in excess of natural levels within 10 meters of the roadbed, beyond which concentrations decline abruptly, depending on traffic density and volume and vehicle speed (Page and Ganje, 1970; Motto et al., 1970; Quarles et al, 1974; Wheeler and Rolfe, 1979; Pierson and Brachaczek, 1976; Mielke et al., 1983; LaBelle et al., 1987). Soil lead concentrations in a sample of city parks have also been reported to be high, ranging from 200 to 3300 ppm (Chow et al., 1975; Zimdahl and Hassett, 1977).

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*Because of the vertical gradient of lead in soil (i.e., lead concentration decreases with soil depth), the techniques used in sampling soils influence the lead concentrations found. In this report, soil lead data refer to the upper 1-5 centimeter layer, which is the most relevant for potential intake by children.

Topsoil lead concentrations around various lead point sources generally decrease exponentially within a 3-10 km radius from 100-60,000 ppm down to background levels (Yankel et al., 1977; Schmitt et al., 1979; Roels et al., 1978; Diemel et al., 1981; Fugas, 1977). However, elevated soil lead levels have even been found at distances exceeding 20-25 km from some smelters (Wixson, 1978). Limited data indicate that lead in soil near primary and secondary lead smelters occurs as lead oxides, sulfide, sulfate, and elemental lead (Olson and Skogerboe, 1975; Corrin and Natusch, 1977).

Much of the lead in the atmosphere is deposited on terrestrial surfaces where it is retained in organic complexes or adsorbed to hydrous iron oxides near the soil surface (CD, p. 7-31). The ability of soil to immobilize lead largely depends on soil pH and organic content (i.e., fulvic and humic substances). Many U.S. soils appear to have a large capacity to bind lead with only a small fraction dissolved in soil moisture where it is available for plant uptake. The environmental impact of lead on the biota of terrestrial and aquatic ecosystems is discussed in Chapter 6 of the CD.

Improvements in air quality do not necessarily involve reductions in soil lead at the same rate. The decontamination of the environment is a very complex and slow process, particularly for soil lead which has been shown to remain at elevated levels several years after lead manufacturing plants have ceased operating (Prpic-Majic et al., 1984). While lead in soil is relatively immobile, it still may enter the food chain, be ingested directly, be mobilized by mechanical forces such as gardening, or may be redispersed into the air and resuspended into water by wind scattering of soil particles and dust.. Consequently, populations living in areas with historical accumulations of lead in soil may continue to be exposed to lead for indefinite periods despite effective controls of atmospheric emissions.

C. Lead in Dust

Dust is a normal component of the home (i.e., "house dust") as well as the outdoor environment where it can be found on sidewalks, playgrounds, driveways, and other hard surfaces. Anthropogenic materials deposited on these outdoor surfaces will be referred to as "street dusts". In addition, the very top layer of soils (including leaf litter) to which people, particularly children, come in direct contact, are considered in the criteria document to be "soil dusts".

Both house dust and street/soil dust contain lead from atmospheric deposition, "natural" soil, and paint chips. As with roadside soil, significant correlations between lead concentrations in street dust and the proximity and density of traffic have been found (Rolfe et al., 1977; Lau and Wong, 1982). Concentrations of lead in dusts on playgrounds, streets, and soil surfaces also increase with higher air lead concentrations and with proximity to stationary lead sources (Roels et al., 1980; Brunekreef et al., 1981; Yankel et al., 1977). As summarized by Nriagu (1978), street dusts from different U.S. cities contained between 300 and 18,000 ppm lead. A survey of street dusts in 77 midwestern cities showed an average lead content of 1,636 ppm in residential neighborhoods and 2,413 ppm in commercial and industrial areas (NAS, 1980). Limited chemical analyses of roadside soils and dusts from U.S. cities found lead predominately as sulfate, along with minor amounts of oxide and halide salts (Olson and Skogerboe, 1975; Corrin and Natusch, 1977). Pavement dust, street dust, gutter debris, and household dust samples contain mostly large lead particles ranging between 40 and 1000 μm (Pierson and Brachaczek, 1976; Sturges and Harrison, 1985; Duggan and Inskip, 1985). Higher lead concentrations are usually found in the smaller sized fractions. The concentration in a sample of street dust sieved at 100 μm, for example, is usually at least two to four times greater than that in the same sample sieved at 1,000 μm (Duggan and Inskip, 1985) and 76% of the composite weight of house dust was measured in particles smaller than 149 μm (Que Hee et al., 1985). This is significant given that the bioavailability of soil lead tends to increase with decreasing particle size (Barltrop and Meek, 1979). In addition, preliminary work indicates that smaller particles (<100-250 μm) readily adhere to fingers and are thus likely to be ingested (Duggan and Inskip 1985; Que Hee et al., 1985).

Unlike lead that is incorporated into soil, lead in surface dusts, both indoors and outdoors, is mobile and can be expected to respond over time to a much greater degree to changes in atmospheric lead emissions. Airborne lead deposited on streets, sidewalks and driveways is subject to distribution by wind and water. Windblown particles associated with dust are apt to be redeposited within the urban environment by the complex wind currents caused by buildings and street canyons. Following the installation or supplementation of emission controls at lead point sources, and significant improvements in air lead quality, significant drops in concentrations of lead in playground dust, surface dust on soil, and in house dust have been observed within 1 to 2 years (Morse et al., 1979; Prpic-Majic et al., 1984). Another study found a weak correlation between dust lead samples collected two months apart in the same houses in Wales (Davies et al., 1985). The authors noted that this is consistent with the suggestion that external influences, e.g., soil, are important for dust lead and differences in weather or season could influence the amount of soil brought into the house and thereby cause short-term changes in dust composition. This illustrates again the potential responsiveness of house dust lead levels to changes in air quality.

There is some evidence to suggest that this same relationship applies in non-point source areas and that lead in surface dusts should decline as a result of the decline in gasoline lead emissions. A survey of rainwashed areas in the U.K. and New Zealand found that the normal acidity of rain (pH between 4 and 5) was insufficient to completely dissolve and transport lead particles, and that residue near streets poses a health hazard to children who are prone to ingest street dusts (Day et al., 1979). However, Laxen and Harrison (1977) found that a light rainfall (2 to 3 mm) is sufficient to remove 90% of the lead from the road surface, mainly to surrounding soil and to waterways. Furthermore, based on deposition fluxes around a major roadway over a year and concentrations of lead in the road drainage water, Harrison et al. (1985) estimated that removal of lead in runoff waters exceeds that deposited on the roadsides. The rate of removal of lead from street dusts is also dependent on the frequency and efficiency of street cleaning operations. It appears reasonable to expect that with declining input, lead concentrations in street and household dusts will also drop due to precipitation, wind, street and house cleaning.

Surveys of a diverse set of homes indicate a wide range of house dust lead levels between 10 and 35,000 ppm (EPA, 1977; Harrison, 1979; Angle and McIntire, 1979; Thornton et al., 1985; Clark et al., 1985) and as high as 100,000 ppm within 2 km of a smelter (Landrigan et al., 1975). Lead levels in house dust can vary considerably depending on house cleaning practices, as demonstrated by Charney et al., (1983), as well as on the presence and condition of lead-based painted surfaces, the presence of cigarette smoke, the amount of dust and soil blown into or carried into the house on clothing and shoes (especially on those occupationally exposed to lead) and pets, indoor sources of lead other than paint (e.g., soldering), the permeability of the home to outdoor air (which can vary with season), and outdoor concentrations of air lead. Lead concentrations in house dust are also dependent on other constituents in the dust and it is therefore important to consider the total amount of leaded dust as well as dust lead concentration.

To assess the impact of atmospheric lead on children's total exposure, it is necessary to estimate the contributions of different air lead levels to outdoor and indoor soil/dust lead levels as well as the amount of dirt a child may ingest, both inadvertantly and deliberately. These issues are addressed in the modeling approaches presented in Sections IV and V.

D. Lead in the Diet

The ingestion of food is a major component of most individuals' total lead uptake, although the relative contribution is a function of their age, and the size and type of diet. The occurrence of lead in the diet may be a result of a) natural sources of lead; b) deposition of airborne lead particles onto crops, forage, feed, soils, and water; and c) the harvesting, processing, transportation, packaging, preparation, and storage of food during which lead can be introduced at every stage either by atmospheric deposition or through metallic contamination, particularly from plumbing or solder in cans.

Recent studies on lead content of various foods, both before and after processing, packaging, and preparation, and on food consumption patterns in the U.S., provide information on dietary lead intakes for different populations (Beloian and McDowell, 1981; Wolnik et al., 1983; National Food Processors Associations, 1982; Pennington, 1983; U.S. FDA, 1983, 1984). Using these data for 1982-83, a "Multiple Source Food Model" was developed in the CD that apportions lead in "typical" child and adult diets to the following sources: natural soil lead, direct atmospheric lead added to food crops before harvest and during processing, indirect atmospheric lead that has been incorporated in soils, lead in solder from food cans and drinking water, and lead whose origin cannot be determined at present (CD, Section 7.3.1.2). The model was validated by using the same parameters along with more recent food data from 1984 and 1985 to successfully predict continuing declines in dietary lead intake. For example, dietary lead intake for a 2-year old child has dropped from about 45-50 µg/day in 1978 to an estimated 13.1 µg/day in 1985; comparable declines (from 45-50 µg/day to 15-20 µg/day) have been seen in different adult populations since 1982 (Flegel et al., 1988.) Even greater reductions had been achieved in the early to mid-1970's (CD, p. 7-49). These trends are attributable to the drastic reduction in gasoline lead emissions and the voluntary phaseout of lead-soldered cans by U.S. manufacturers since the 1970's. This downward shift is expected to continue up until the early 1990's as lead from both these sources drops further. The methods by which estimates of dietary lead intake from the Multiple Source Food Model are derived and applied to modeling total lead exposure are discussed in Appendix A and Section V.

E. Lead in Water

Lead is a natural, usually very minor, constituent of surface and ground waters. Atmospheric lead can enter aquatic systems through direct fallout or in surface runoff as suspended particles or adsorbed to soil particles. Under most conditions (pH, temperature, alkalinity), lead forms insoluble salts and precipitates to sediments, which probably accounts for the low lead content of U.S. surface water supplies; the average concentration ranging between 3 and 4 micrograms Pb per liter water (µg/L) (NAS, 1980).

In contrast, lead levels in household, school, and office building drinking water can be much higher due to plumbing corrosion and subsequent leaching of lead, ranging between 10 and 30 µg/l on average. The combination of corrosive (i.e., soft or acidic) water and lead pipes or lead soldered joints in distribution systems or houses creates localized zones of high lead concentrations up to 380 μg/l (Worth et al., 1981). The combination of new solder and corrosive water can result in concentrations commonly over 1000 mg/L. In general, levels are highest in samples of hot and/or stagnant "first draw" water.

Drinking water is a major source of lead exposure for many infants while they are dependent on baby formulas during their first year, as well as for young children. The 1986 Safe Drinking Water Amendments banned the use of lead solder or flux and lead-bearing pipes and fittings, to be implemented and enforced by the States by June 1988. In August 1988, EPA's Office of Drinking Water proposed revising the existing lead standard of 50 μg/L to 5 μg/L, measured at the entry point to the distribution system or the treatment plant, with a "no-action level" average of 10 μg/L, measured at the home tap.

Estimated contributions that natural, atmospheric, and solder lead make to total lead exposure via drinking water and total diet are addressed in the modeling approaches presented in Sections IV and V.

F. Lead in Paint

Ingestion of lead-containing paint is considered to be the most frequent cause of severe lead intoxication among children (Chisolm, 1984; CDC, 1985). Significant correlations have been found between the quantity and condition of lead painted surfaces in homes and blood and fecal lead levels in inner city children (Urban, 1976; CD, pp. 11-156 to 11-160). Significant differences in blood lead levels have been found in relation to housing condition among children as young as nine months, with highest levels in children living in deteriorating pre-World War II housing, intermediate levels in well-maintained and rehabilitated older housing, and lowest levels in children in public housing and newer units free of lead paint (Clark et al., 1985). Other investigations indicate that in addition to peeling lead-based paints, "intact" lead-based paint contributes to elevated blood lead levels in children via chalking or weathering (Gilbert et al., 1979; Galke et al., 1975) or can be exposed to children who chew or gnaw upon the surfaces. Further risks are associated with conventional paint removal techniques (i.e., sanding, burning heat guns) which can generate concentrations of fine lead dust up to 100-100,000 μg/m^3 (NIBS, 1988; Inskip and Atterbury, 1983).

Under the 1971 Lead-Based Paint Poisoning Prevention Act, which has since been amended, the Department of Housing and Urban Development has regulatory and research responsibilities for eliminating the hazards of lead-based paint poisoning in federally funded housing. In 1977, the Consumer Product Safety Commission banned household paint (including toy and furniture paint) containing more than 0.06% lead as hazardous. This was an important step but it does not affect hazards posed by lead-based paints already present on old housing surfaces. Prior to 1940, some interior paints contained more than 50% (500,000 ppm) lead. Use of lead pigment paints declined slowly between 1940 and the late 1960's after which it declined at a rapid rate (NAS, 1972). Pope (1986a) estimates that in 1980, the interior and/or exterior surfaces of between 21.5 and 47.3 million households in the U.S., mostly built before 1960, contained greater than 0.7 mg/cm^2 lead in painted surfaces, a level considered hazardous to young children by the Centers for Disease Control (CDC). Between 6.2 and 13.6 million children under the age of 7 are estimated to have resided in lead-based painted housing in 1980 (Pope, 1986a). Even if coated with low-level leaded paint, the underlying layers of lead-based paint in older homes represent a large reservoir of lead exposure in children, particularly if there is peeling paint, broken or cracked plaster, or holes in the walls. The number of lead-based painted homes with these deteriorating conditions (i.e., "unsound") is estimated to have been between 800,000 to 2.9 million in 1980 with approximately 235,000 to 842,000 children under age 7 living in such homes (Pope, 1986a).

Lead-based paint continues to be the major source of high-dose lead exposure and symptomatic lead poisoning for children in the U.S. (Chisolm, 1971; CDC, 1985), and it appears that exposure to lead-based paint will continue to be a problem for decades to come (Schneider and Lavenhar, 1986). Between 1973 and 1980, only 10% of pre-1940 housing units and .5% of housing units built in the 1940's had been removed from our nation's housing stock by demolition, disaster, or by other means such as conversion to commercial space (Bureau of the Census, 1983). Less than half of these removed units were located in central city areas where lead poisoning is most prevalent. In addition, poor urban families often have no choice but to live in poorly maintained older housing, given that the vast majority of new housing units created from 1973 to 1980 is located outside of central city areas and that acute shortages of modern and lead-free, low income rental units exist in many cities (Farfel, 1985). Many rural families are also afflicted with a lead paint risk since about 41% of rural housing is "substandard" (ATSDR, p. VI-15).

High exposures to children living in older housing with flaking or intact lead-based paint will not be significantly influenced by any changes in atmospheric lead emissions. Children with pica for paint chips or others living in deteriorating lead-based painted homes who are excessively exposed to paint lead-contaminated dust through normal hand to mouth activity cannot be expected to be protected by any lead NAAQS no matter how stringent. For this reason and because exposure to paint lead is so highly variable, no attempt will be made here to quantitatively estimate exposures of children who are excessively exposed to lead-based paint under various air lead levels, as is done for other exposure media. Some of the available data, although sparse, are discussed at the end of Appendix A to demonstrate how some estimates could be generated. Despite the small amount of data, and the great variability in paint lead exposures, it is clear that any exposures and blood lead levels predicted for children under various air lead levels using the approaches in Section III will be significantly higher for children with high paint lead exposure. Comprehensive plans for safe and effective removal or containment of lead painted surfaces from old housing need to be developed and implemented, otherwise pediatric plumbism will persist until much of the present housing stock is completely rehabilitated or demolished (Chisolm, 1986). Preventing excessive exposure to existing sources of lead in and around housing must be addressed by an appropriate combination of legislation, housing code inspection and enforcement, financial incentives, parental education, and other programs.

III. ESTIMATING LEAD EXPOSURES AND BLOOD LEAD LEVELS

A. Alternative Modeling Approaches

To assess the health risks associated with alternative air lead standards, it is necessary to estimate the blood lead (PbB) levels that would be distributed in the population(s) of concern under various air lead concentrations. [The amount of lead measured in whole blood is an index of the rapidly diffusible fraction of the total body burden of absorbed lead and is generally used as the dosage, or index of exposure, in investigating the various human health effects associated with lead.] Lead exposure occurs through multiple media but unfortunately, longitudinal assessments of simultaneous multi-media exposures have not yet been made. Potentially useful longitudinal analyses are now being carried out as part of the Cincinnati Lead Program Project (Clark et al., 1987). Three approaches are examined here that can be used to estimate or predict the impact of inhaled and ingested lead aerosols and compounds on the body burden of lead as indexed by blood lead. These approaches are presented in detail in Sections IV and V for consideration for using one, two, or all three in assessing the protection provided by alternative lead NAAQS.

The first approach, discussed in Section IV, is to use measured rates of absorption, or "uptake" of lead through different pathways (e.g., inhalation and ingestion) from experimental studies together with available mathematical ("biokinetic") models from lead balance studies to project either total body burden or the amount of lead in any of the presumed "physiological" kinetic compartments (e.g., blood, soft tissue, bone) at any time.

The second approach, referred to as an "aggregate" model, is to directly apply a mathematical relationship between air lead and blood lead derived from community epidemiological studies that reflects both direct inhalation exposure and indirect exposures via secondary deposition processes. The third approach, referred to as a "disaggregate" model, is a hybrid of the first two in which separate empirical relationships between blood lead and lead intake from lead in air, food, water, dust, and soil, available from experimental exposure and epidemiological studies of different populations, are applied to lead concentration estimates in different media. The nature of the relationships detected between lead concentrations in blood and various environmental media, and the merits and limitations in using empirical relationships to represent lead exposure through multiple pathways in these latter two approaches, will be discussed in Section V.

B. Calculating Blood Lead Distributions Around Predicted Means

Because most of the data input to these models are generally average estimates, all three models estimate PbB levels in terms of population mean levels. The distribution of PbB levels in U.S. children is broad because there is a distribution of exposures those children face, a distribution of behavior patterns that affect the uptake of that exposure, and a distribution of biological absorption and excretion rates. For purposes of setting a lead NAAQS, it is necessary to determine the blood lead distribution across a defined population group corresponding to a given mean PbB for that group so that, for example, percentages of individuals exceeding PbB levels of concern can be ascertained. Determining such distributions is an attempt to account for the significant behavioral and biological variability within populations. It is the group of individuals with the potentially greatest adverse response to any given lead exposure that are of greatest concern in establishing a primary lead NAAQS.

Consistent with measurements of other metals in tissues of human populations, the distribution of PbB levels for any relatively homogeneous population closely follows a lognormal distribution (CD, Section 11.3.4). A lognormal distribution is completely specified by its geometric mean and geometric standard deviation. It is possible, therefore, to calculate percentiles of a blood lead distribution (e.g., median or 99 percentile) around a mean PbB level by using the geometric standard deviation (GSD) of that population's blood lead distribution. Several epidemiological studies have indicated that the log values of measured individual PbB levels in a uniformly exposed population are normally distributed with a variation, including analytical variation, ranging between 1.3 and 1.4, when expressed as a GSD (Tepper and Levin, 1975; Azar et al., 1975; Billick et al., 1979). The NHANES II study provides the best available data on nationwide blood lead levels in terms of quality control and sample size. In regression analyses of these data, Schwartz (1985a) estimated a GSD of 1.428 for young children aged 6 months to 5 years, after removing the variance in PbB levels attributable to air lead exposure by adjusting every individual's PbB level to what it would be at zero air lead, while allowing for variations in background non-air lead exposure.

After excluding from the analysis children with PbB levels above 40 μg/dl, who would not be expected to be substantially affected by changes in the lead NAAQS (for example, because of higher than average pica activity or excessively high exposures to lead in paint), Schwartz (1985b) reapplied the same technique and calculated a GSD of 1.419. Thus, a GSD without attribution of any source of lead exposure except gasoline lead and industrial air lead emissions may be taken as a rounded-off value of approximately 1.42 for the NHANES II population of children (CD, p. 11-31).

Given that the focus of this exposure analysis is on populations living near lead point sources where blood lead variability may be different compared to the total mix of U.S. children, a separate assessment of blood lead GSD's for such populations is presented here. Published statistics of PbB samples near various lead point sources have been assembled and analyzed in Cohen (1986) and Marcus (1988a). The GSDs calculated from these point-source/blood lead surveys are listed in Table 3-1, along with the NHANES II GSD for children. To different degrees, the primary origin of non-biological variation in lead exposure for these point source populations was the smelter, especially in the Roels and Yankel studies conducted when smelter emissions were considerably higher. It is possible that children with exposure to a single, intense source of lead may have relatively low variability (essentially only due to intrinsic biologic variability) with a high mean blood lead. The most recent study indicates a larger variance at relatively lower exposure levels near the Montana smelter surveyed in 1983, suggesting that multiple origins with separate variability may be present rather than a single predominant source of variability in lead exposure (Marcus, 1988a).

The selection of a GSD value to model populations living near various lead point sources in the future depends on assumptions regarding variance in exposure in the future. Perhaps the most relevant data are from the 1983 E. Helena, Montana study since they reflect the most contemporary conditions. It can be expected that even for this population, however, total lead exposure will have dropped substantially by 1990, the starting point of our exposure analyses. This is attributable to recent and continuing downward trends in lead levels in canned foods, the nationwide ban on lead solder in new construction and plumbing repairs, the lead-in-gasoline phasedown, and perhaps increasing public awareness regarding the dangers of lead exposure and effective avoidance measures. All of these changes should result in not only lower mean baseline

TABLE 3-1. COMPARISON OF ESTIMATED GSDs ACROSS SEVERAL STUDIES

Population/Reference	Mean PbB ($\mu g/dl$)	Estimated GSD[1]
NHANES II - total mix of children 1-5 years old (Schwartz, 1986b)	16.0	1.42
11-year olds living near Belgian primary lead smelter (Roels et al., 1980)	21.7	1.29
1-9 year olds living near Idaho primary lead smelter (Yankel et al., 1977)	56.5	1.32
1-5 year olds, closest to 3 non-ferrous smelters in U.S. (Hartwell et al., 1983)	15.6 (median)	1.39
1-5 year olds, living near Montana primary lead smelter (CDC, 1983)	9.4	1.53
1-5 year olds living in 3 Missouri smelter towns (Baker et al., 1977)	16.2	1.57

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[1] GSD calculations described in Cohen (1986) and in Marcus (1988a).

PbB levels, but perhaps more importantly, in fewer high level exposure situations and therefore less variance in PbB levels than existed in 1983. Quantifying such an effect to estimate a future GSD value is difficult. It is likely, however, that future reductions in baseline "non-point source" lead exposures will result in a reduction in exposure variance so that the blood lead GSD will be more reflective of intrinsic biologic variance, such as that found in the Roels and Yankel studies. Although these latter studies were conducted during times of much higher exposures, the exposures were dominated by high smelter emissions and soil/dust contamination, resulting in relatively low PbB variability. For purposes of modeling future exposures among children living near different lead point sources, a range of GSD values can be considered: a lower bound of approximately 1.30 derived from the Yankel et al. and Roels et al. studies and an upper bound GSD value of 1.53 derived from the 1983 CDC study of E. Helena, Montana children. The midpoint of this range is 1.42, which coincidentally is identical to the NHANES II GSD estimate. Until additional data are available, a range of 1.30-1.53 will therefore be assumed for children living near lead point sources as a reasonable range of GSD values, and the midpoint of 1.42 will be assumed as a reasonable best estimate. In the case study exposure analyses, the larger and more conservative GSD of 1.53 will be tested in sensitivity analysis.

Since blood lead estimates will also be made for middle-aged men, GSD values for adult blood lead distributions are needed. There is little information on adult blood lead GSDs from point source studies. The most complete data base is clearly from NHANES II, which has been analyzed in the CD to yield a range of GSD values, depending on race and place of residence, between 1.34 and 1.39 for adult women and between

1.37 and 1.40 for adult men (CD, Table 11-9). Given the uncertainties in extrapolating these ranges to adult populations living near point sources under future exposure scenarios, a midpoint value of 1.37 will be chosen as a reasonable estimate for both adult men and women.

Analytical variation which exists in any measurement has an impact on the bias and precision of statistical estimates such as the GSDs derived from the studies discussed here. For this reason, it is important to recognize the magnitude of analytical variation in blood lead measurements due to measurement variation (i.e., between measurements run at the same time) and variation created by analyzing blood samples at different times (CD, p. 11-29). Using GSDs adjusted in such a way, although providing a more accurate characterization of a given blood lead distribution, may not be appropriate for this assessment because the health effects studies that will be used to define dose-response relationships did not correct their PbB measurements for analytical variance. These studies, like NHANES II and the point source epidemiological surveys, generally employed the best available measurement techniques and quality control. Using "corrected" GSDs to predict the distribution of children's PbB levels around a mean population PbB, and then matching those predicted PbB levels with "uncorrected" PbB levels derived from health studies in order to assess the risks associated with that population mean PbB, would result in a somewhat biased assessment. Therefore, the range of GSDs presented above that are not corrected for analytical variance will be used in further calculations.

Table 3-2 presents blood lead distributions associated with sample population mean PbB levels calculated using the GSD values derived above.

These distributions were calculated using the formula given to describe a lognormal distribution (Yankel et al., 1977): $PbB = GM(GSD)^z$

where:

GM = geometric mean PbB

GSD = geometric standard deviation

z = the number of standard deviations

PbB = value of PbB at z standard deviations

TABLE 3-2. ILLUSTRATIVE BLOOD LEAD DISTRIBUTIONS ASSUMING DIFFERENT MEAN BLOOD LEADS AND GEOMETRIC STANDARD DEVIATIONS

GSD	Geometric Mean PbB	Percentile			
		90th	95th	99th	99.5th
1.37	4	6.0	6.7	8.3	9.0
	5	7.5	8.4	10.4	11.3
	6	9.0	10.1	12.5	13.5
	7	10.5	11.7	14.6	15.8
1.42	4	6.3	7.1	9.0	9.9
	5	7.8	8.9	11.3	12.3
	6	9.4	10.7	13.6	14.8
	7	11.0	12.5	15.8	17.3

IV. INTEGRATED LEAD UPTAKE/BIOKINETIC MODEL

The "uptake/biokinetic" modeling approach attempts to account for the following: a) the amount of lead in the body at any one time is the product of dynamic interactions of partially offsetting processes of absorption, distribution, storage, mobilization, and excretion; b) these processes vary with the route and rate of exposure, a person's age, nutritional and health status, and baseline exposure; c) uptake from all sources by all absorption routes can be separately modeled, thus providing an estimate of the relative importance of atmospheric lead exposure, either directly or indirectly, to total body burden; and d) past and future trends in environmental lead levels due to different control efforts and regulations are important determinants of projected exposures.

There have been several studies measuring the intake, uptake, and metabolism of lead in adult male volunteers, from which balance schemes have been constructed (Kehoe, 1961; Chamberlain et al., 1978, Rabinowitz et al., 1976, 1977). By making estimates about lead intakes and various metabolic factors, balance schemes can be constructed for different groups with different exposures to lead to assess the importance of specific exposure factors under variable conditions (e.g., alternative lead NAAQS, phaseout of lead in soldered cans and in gasoline). Pre-school age children (≤ 6 years old), pregnant women (as exposure surrogates for the fetus), and adult men (40-59 years of age) are specified in the CD as particularly sensitive to lead. Of these groups, children between 2 and 3 years old exhibit, in general, the highest blood lead levels (Mahaffey et al., 1982; Billick et al., 1979), most likely due to their greater hand-to-mouth activity as well as to various metabolic processes (Harley and Kneip, 1985). To predict exposure impacts of alternative air lead standards, model balance schemes have been devised for children aged 0-6 years and are discussed below.

A lack of adequate biokinetic data for pregnant females prevents such modeling for the fetus. In addition, accurate biokinetic modeling of the sensitive population of adult males would require at least 40 years of cumulative exposure inputs. Such uptake modeling would be difficult given the great variability in exposure histories among U.S. adults and the paucity of data. Exposure modeling for purposes of risk estimation for adults, discussed in Section V.A., will rely on combining air: blood lead "disaggregated" relationships with estimated baseline "non-air" blood lead levels.

A discussion of lead's absorption, excretion, retention, and distribution within the body under different exposure and physiological conditions is necessary background to the uptake/biokinetic model, and is provided in Chapter 10 of the CD. As will be further discussed, application of this or any lead exposure model must recognize the limitations of the available data and the significant variability among populations in their behavioral, exposure, and physiological characteristics. For present purposes, the children to be considered in the model do not include the whole U.S. population; they comprise groups with contrasting exposures to lead, some of them extreme, in order to illustrate the variations in lead exposure and absorption around lead point sources under different scenarios. Section IV.A presents methodologies to estimate average daily lead uptake from all exposure sources under alternative air lead levels. Section IV.B presents methodologies to relate these estimates of lead uptake to average, steady-state blood lead levels. Although this approach predicts hypothetical outcomes and the absolute numbers should not be used uncritically, the model does strive to use the available data, with all its limitations, to the fullest extent possible to provide a useful tool in eventually distinguishing the health impacts of alternative lead NAAQS.

A. Estimates of Lead Uptake

The method employed to estimate the degree to which each environmental source of lead contributes to a child's total daily lead uptake is based on the distribution of ambient air lead levels that would be allowable or expected under each standard considered. Probable exposure conditions with respect to other exposure media such as food and dust, and average biological absorption rates for each exposure route are also specified. The method consists of a four-step process:

1) definition of ambient concentrations of lead for major exposure sources (i.e., air, food, water, soil, dust);

2) determination of daily lead intake according to the relationship:

$$I_i = C_i \cdot [Pb]_i$$

where I_i is the daily lead intake from source i, C_i is the ingestion or inhalation (i.e., consumption) per day of each lead source i and $[Pb]_i$ is the concentration of lead in each source i;

3) calculation of the amount of lead absorbed from each exposure source i:

$$U_i = I_i \cdot A_i$$

where U_i is lead uptake for each exposure source i, I_i is the daily lead intake from each source i, and A_i is the percent absorption of lead, via the appropriate exposure route for the particular source; and

4) calculation of the total lead uptake from all sources, U_μ:

$$U_\mu = \quad (I_i \cdot A_i)$$

The way daily lead uptake would be calculated is illustrated in Table 4-1 for 2-year old children living near (within 2-5 km) a lead point source. Details of the model parameters and the line-by-line estimates, along with

TABLE 4-1. ESTIMATED 1990 AVERAGE LEAD INTAKE AND UPTAKE IN 2-YEAR OLD CHILDREN UNDER AVERAGE AIR LEAD LEVELS[1]

	0.25 μg/m³	0.5 μg/m³	0.75 μg/m³	1.0 μg/m³	1.25 μg/m³	1.5 μg/m³
1. Outdoor air lead (μg/m³)	0.25	0.5	0.75	1.0	1.25	1.5
2. Indoor air lead (μg/m³)	.08	0.15	0.23	0.3	0.38	0.45
3. Time spent outdoors (hours/day)	2-4	2-4	2-4	2-4	2-4	2-4
4. Time weighted average (μg/m³)	.09-.11	.18-.21	.27-.32	.36-.42	.45-.54	.54-.63
5. Volume of air respired (m³/day)	4-5	4-5	4-5	4-5	4-5	4-5
6. Lead Intake from air (μg/day)	0.4-0.6	0.7-1.1	1.1-1.6	1.4-2.1	1.8-2.7	2.2-3.2
7. % deposition/absorption in lungs	42	42	42	42	42	42
8. Total lead uptake from lungs (μg/day)	0.2-0.3	0.3-0.5	0.5-0.7	0.6-0.9	0.8-1.1	0.9-1.3
9. Dietary Lead Consumption (μg/day)						
a) from solder or other metals	5.6	5.6	5.6	5.6	5.6	5.6
b) atmospheric lead	1.2	1.2	1.2	1.2	1.2	1.2
c) natural lead, indirect atmospheric, undetermined sources	3.6	3.6	3.6	3.6	3.6	3.6
10. % absorption in gut	42-53	42-53	42-53	42-53	42-53	42-53
11. Dietary lead uptake (μg/day)	4.4-5.5	4.4-5.5	4.4-5.5	4.4-5.5	4.4-5.5	4.4-5.5
12. Street dust/soil lead (μg/g)	181	308	436	563	691	818
13. Indoor dust lead (μg/g)	271	482	693	904	1115	1326
14. Time weighted average (μg/g)	241-256	424-453	607-650	790-847	974-1044	1157-1241
15. Amount of dirt ingested (g/day)	.08-.135	.08-.135	.08-.135	.08-.135	.08-.135	.08-.135
16. Lead intake from dirt (μg/day)	19.3-34.6	33.9-61.2	48.6-87.8	63.2-114.3	77.9-140.9	92.5-167.6
17. % dirt lead absorption in gut	25	25	25	25	25	25
18. Lead uptake from dirt (μg/day)	4.8-8.6	8.5-15.3	12.1-21.9	15.8-28.6	19.5-35.2	23.1-41.9
19. Total lead uptake from lung and gut (μg/day)	9.4-14.4	13.2-21.3	17.0-28.1	20.8-35.0	24.7-41.8	28.4-48.7

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[1] Assumptions and calculations are described in Appendix A by row number. Refers to children living near one or more lead point sources and unaffected by lead paint. Children living in typical urban or rural environments remote from lead point sources can be modeled using the same uptake calculations and substituting parameter values where differences are noted in Appendix A.

limitations of the available data base, are discussed in Appendix A. For comparative purposes, children living in areas generally unaffected by stationary lead emissions can also be modeled using most of the same parameter values; exceptions (indoor/outdoor particled penetration rates, respiratory deposition rates) are noted in Appendix A. Dust and soil lead levels associated with given air lead concentrations would differ substantially between point and non-point source areas; this too is briefly discussed in Appendix A. The following points are important to consider with regard to the uptake calculations in the table: 1) the calculations do not represent population-wide exposure scenarios under alternative standards, only exposure for the specified air lead concentrations. Ambient lead levels around a point source meeting a given lead NAAQS vary depending on distance, topography, local meteorology related to pollutant transport and diffusion, plant operating parameters, emissions controls, particle sizes and deposition rates, etc. Air quality dispersion modeling that accounts for these factors will be used in the actual case study analyses to assign air lead concentrations under alternative standards to different "receptor" points where people live around the lead point sources; 2) the estimates in the table apply to 2-year old children to illustrate the period of maximum exposure. Blood lead levels estimated by the biokinetic model (discussed below) account for accumulating exposure throughout childhood, not just a single year. It is necessary, therefore, to make age-specific exposure estimates that reflect changes in behavior and metabolism as a child ages. Age-specific exposure estimates are also discussed in detail in Appendix A; and 3) the general types of exposure environments considered here do not include older homes with lead-based paint hazards. Such conditions are discussed at the end of Appendix A to illustrate the severity of lead-paint hazards compared to other exposures.

B. Uptake and Blood Lead Concentration

To estimate blood lead (PbB) levels under different exposure scenarios, several different kinds of studies can be used to derive a relationship between absorbed lead (or lead uptake) and blood lead. Available studies include population surveys in which the blood lead concentration of individuals or groups is correlated with measured lead concentrations in air, food, water, soil, or dust; experiments in which volunteers are exposed to controlled air lead concentrations and their PbB levels measured, and; lead balance studies of individuals with measured lead intakes. The most relevant experimental studies including, for comparative purposes, those on adults, are discussed here along with descriptions of the analyses used to derive uptake/PbB relationships. Relationship between blood lead and environmental lead concentrations from population surveys are discussed as part of the aggregate and disaggregate models in Section V.

1. Dietary Lead Ingestion Studies in Infants

Studies that have measured dietary lead intake concurrently with PbB levels among infants and toddlers are compared in the criteria document (CD, Table 11-49). Although precise estimates of dietary lead consumption can not be made, these studies (U.K. Central Directorate, 1982; Lacey et al., 1985; Sherlock et al., 1982; Ryu et al., 1983) have several advantages: 1) careful control and analysis of dietary intake; 2) minimal lead exposure to sources other than the diet as the study population (infants) is relatively immobile; and 3) intake/blood lead relationships can be used to estimate the impact of total lead exposure (i.e., from all sources) on blood lead because tracer studies (Chamberlain et al., 1978; Rabinowitz et al., 1976) show no differences in the distribution of lead to tissues whether taken up from lung or gut.

Both the U.K. Central Directorate and the Sherlock et al. studies involved infants with relatively high PbB levels and high intakes. It is well documented that the relationship between PbB and lead uptake from any source is approximately linear at low intake levels but "flattens out" at high intake levels (CD, Sections 11.2 and 11.3). Because the PbB levels (< 20 µg/dl) and lead intakes of the Ryu infants are more relevant, the criteria document concludes that the slope from this study (0.16 µg/dl per µg/day lead intake) is the best available estimate (CD, p. 11-129).

The infants in the Ryu et al. (1983) study absorbed substantial quantities of lead from formula or whole milk in lead-solderd cans. Even lead at much lower levels from formula in glass bottles achieved an apparent blood lead equilibrium postnatally. Reanalyses of the Ryu et al. data by Marcus (1989) suggested a larger slope, about 0.24 µg/dl per µg/day lead intake in formula and milk.

Using non-linear and piecewise linear models to examine PbB-dietary lead slopes at higher intake levels, Marcus (1989) reanalyzed the Ryu et al. data, as well as data on Scottish infants (Lacey et al., 1985) and school children (Laxen et al., 1987) exposed across a wide range of water lead levels. Because the diets among the infants in the Lacey et al. study were predominantly liquid, the derived water lead/blood lead relationship is considered a surrogate to predict the contribution of total diet to blood lead levels. A piecewise linear model fitted the data about as well as a cube-root or square-root model, with a PbB/water Pb slope from the Lacey et al. study on infants of 0.254 µg/dl per µg/L for water lead levels below 16 µg/L, and a much lower slope of 0.0426 above that inflection point; after partialling out the effects of house dust lead, the Laxen et al. school-children had a PbB/water Pb slope of 0.161 µg/dl per µg/L for water lead levels below 15 µg/L, and a 0.0318 slope above that level (Marcus, 1989).

If the slopes from Lacey et al. and Laxen et al. are converted to uptake slopes by assuming 1 L/d water consumption, then the three absorption coefficients (0.24 from Ryu et al., 0.254 from Lacey et al., 0.161 from Laxen et al.) are reasonably consistent for low levels of dietary lead intake. Furthermore, given the differences among the three study populations and their exposure situations, the above results are strikingly consistent in terms of the shape of the dose-response relationship, the location of the inflection point, and the magnitude of the differences above and below that point. The higher slope found for the Lacey et al. infants compared to the Laxen et al. school children is expected given the reduction in lead gut absorption rates with age. The uptake of lead from diet depends not only on the age of the child, but also possibly on the chemical and physical form of the lead, and on other components of diet that affect lead absorption. Much lower coefficients may be appropriate for adults and for older children who only ingest lead with meals; even higher coefficients may be appropriate for lead absorbed by very young children who consume lead between meals and who have nutritional deficiencies that facilitate lead absorption.

The lower slope at high levels of water lead or dietary lead uptake probably represents other factors that reduce absorption, e.g., much of the "excessive" dietary lead intake may occur during meals and is thus much less bioavailable. There is reasonable consistency among these high-uptake slopes: 0.0426 from Lacey et al. infants; 0.0318 from Laxen et al. school children; and 0.032 from the Sherlock and Cools studies for adults (see below). Uncertainty about the change-over between high-absorption and low-absorption cases probably reflects incomplte information about the food and water consumption patterns and other dietary factors. For present-day

U.S. dietary lead exposure, the steeper slopes are likely to be more accurate.

The results from the Ryu et al. and Lacey et al. studies will be used to represent the infant dose-response relationship: the midpoint of the slopes (0.16 and 0.254, i.e., 0.207) estimated for low exposure levels (i.e., < 16 µg/L), and the Lacey et al. slope of 0.026 for higher exposure levels. Assuming the same lead concentration in total diet where the slope flattens out, the water lead "threshold" level from the Scottish studies of approximately 16 µg/L (or 16 µg/Kg) can be converted to a corresponding dietary intake level by multiplying by the average dietary intake for an infant. The CD cites a dietary intake rate of 1.502 Kg/day for a 2-year old child (Table 7-15) and a rate of 1.0 Kg/day will be assumed for the infant. The resulting level of 16 µg/day will be used as the inflection point in applying the slopes from Ryu et al. and Lacey et al. (0.207 and 0.026). Because lead uptake/PbB relationships are of interest, these slopes will be combined with the midpoint of gastrointestinal absorption rates (42-53%) cited for infants in the CD, to illustrate the relationship between blood lead and daily lead uptake for infants at the end of this section. A baseline (i.e., non-air) PbB of 4 µg/dl will be assumed, based on recent studies of PbB levels and lead intake in Boston and Cincinnati infants (Rabinowitz et al., 1986; Succop et al., 1987).

2. Dietary Intake Studies in Adults

The relationship between blood lead and food and/or water lead levels has been examined for adults in both experimental studies in which controlled dietary supplements were administered to volunteers, and duplicate diet studies. Again, several studies indicate non-linear blood lead responses at high exposures but a linear relationship at relatively low intake levels (< 100 µg/day, or < 30 µg/dl PbB).

Two studies identified in Table 11-49 and 11-50 of the CD appear most useful in estimating a dietary lead/blood lead relationship chiefly because of their relatively large sample sizes and control over confounding factors: Sherlock et al. (1982), a duplicate diet study of 31 mothers (and their children) from Ayr, Scotland; and Cools et al. (1976) in which 11 subjects and 10 controls were studied for blood lead response to oral dosages of lead acetate.

The latter study was an extension of the Stuik (1974) experiment, results of which will not be used here because responses were not followed long enough for blood lead to equilibrate. The weighted mean of the slopes reported in the CD for the Sherlock and Cools studies is 0.032 µg/dl increase in blood lead per µg/day intake, which is a factor of 5-6 times lower than the comparable slope estimated for infants at low exposure levels. A gastrointestinal absorption factor of 0.15, cited in the CD for adults, will be applied to this intake slope to derive a blood lead/uptake relationship. A baseline PbB level of 4 µg/dl will be assumed for adults based on projections described in Appendix C. The same pattern of non-linear absorption seen in children will be assumed for adults and a slope of .009 µg/dl per µg/day will be used for levels above approximately 40 µg/day, which corresponds to an inflection point of 15 µg/kg lead in diet, multiplied by an approximate dietary intake of 2.89 kg/day for middle-aged men (CD, Table 7-15).

3. Application of Chamberlain and Heard Analysis of Adult Exposure Data

An attempt to relate blood lead and absorbed lead was made by Chamberlain and Heard (1981) using epidemiological and clinical data on adult men (Williams et al., 1969; Kehoe, 1961; Nordman, 1975; Zurlo and Griffini, 1973; Fugas and Saric, 1979). Total dietary and airborne lead uptake was estimated using calculations similar to those presented in the integrated lead uptake model in Section IV.A. The following assumptions given in Chamberlain et al., (1978) were used: a) a fraction, 0.55, of the uptake becomes attached to red blood cells; b) the biological half-life of lead in blood is 18 days; c) a factor, 1.3, is to be allowed for long-term resorption and re-entry into blood of some of the lead which is stored in bone; and d) the mass of blood is 5400 grams. With these assumptions, Chamberlain and Heard calculated the following relationship:

$$\frac{\Delta \text{ PbB}}{\Delta \text{ Uptake}} = \frac{0.55 \times 18 \times 1.3}{54 \times 0.693} = 0.34 \ \mu\text{g/dl per } \mu\text{g/day}$$

This relationship is comparable to that derived from the Cools and Sherlock studies after gastrointestinal absorption is accounted for. Given uncertainties about some of the studies analyzed by Chamberlain and Heard regarding exposure levels and quality control, and because of the high exposure levels, this slope is presented here only for comparative purposes and will not be used further.

4. Lead Balance Studies: Compartmental Models

To demonstrate a causal relationship between lead in the body and a biological change, it would be ideal to know the amount of lead present at the site and time of the effect. For instance, if lead is suspected to induce neuropsychological changes, a measure of the lead level present in nerve cells when the change occurred would be desirable. Living tissues can rarely be sampled, and data obtained at necropsy cannot reveal the variations in exposure throughout the individual's life. For analytical simplicity, tissues can be grouped together on the basis of lead distribution characteristics and body burdens of lead represented as a limited number of distinct, homogenous, and well-mixed pools or physiological compartments with similar kinetic properties. Mathematical biokinetic models have been fitted to data obtained in long-term balance studies and to lead isotope tracer experiments that estimate the rates of input to, transfer between, and excretion from the different compartments (Rabinowitz et al., 1976, 1977; Batschelet et al., 1979; Bernard, 1977; Mallon 1983; Harley and Kneip, 1985; Marcus, 1985a,b,c). Differences in the predictive lead models have been discussed by Bernard (1977), Batschelet et al. (1979) and Hammond et al. (1981). Models are shown schematically in the CD (Figures 10-3 and 10-4).

The choice of pools or level of aggregation in a model depends on the available experimental data, the appropriate time scale, and the predictive uses of the analysis, and is a compromise between accuracy and simplicity. For example, blood can be broken down into plasma and erythrocytes and then further into plasma protein-bound lead, diffusible lead, extracellular fluid lead, and erythrocyte proteins. Soft tissues can be broken down into brain (hippocampus, medulla, etc.), kidneys, liver, hair, and so on. The hard tissue pools can be separated into compact (cortical) and cancellous (trabecular) bones, and teeth; within each of these, separate components may be needed to model distinct diffusion time scales (Marcus, 1985a,c). Because the skeletal system in young children is rapidly developing and is both large and kinetically active, it is especially important to model bone lead in children. Until such refinements are further tested, a relatively simple 3 - 5 pool model provides an adequate framework for predicting lead distributions in children.

These models are helpful in predicting total body burden or equilibrium levels of lead over time in any of the presumed kinetic compartments under different exposure conditions. The basic assumption of the models is that the mass of lead in each of the compartments changes according to a system of coupled first-order linear differential equations with constant fractional transfer rates. Such models predict that when the lead intake changes from one constant level to another, there is a directly proportional change in the mass of lead in each compartment and the attainment of a new equilibrium. The rates and magnitudes of these changes are dependent upon the rates of lead flux in the tissues and can theoretically be calculated from a compartmental model of the appropriate parameters. Support for a first order kinetic model for lead metabolism is demonstrated by calculations, using first order models of soft tissue and bone concentrations of lead, and other elements (calcium strontium, radium), that fit human measurements as well as by using more complex models (Johnson and Myers, 1981; Mallon, 1983).

A four-compartment biokinetic model of lead metabolism has been developed from data obtained in controlled single dose and chronic lead exposures of infant (9 months) and juvenile (22 months) baboons (Mallon, 1983; Kneip et al., 1983). Dynamic blood measurements and steady state blood and organ lead measurements were closely fitted to predict concentrations of lead in blood, liver, and kidney, and bone (the four compartments in which 95% of total body lead is contained) (Heard and Chamberlain, 1984). Human metabolism and growth patterns were applied in a computer simulation of the model that was then successfully validated using human autopsy data.

The model parameters were revised using measured metabolic data for each organ (e.g., bone turnover rates) for children and were used to simulate organ lead burdens and concentrations in children with constant lead exposures from birth (Harley and Kneip, 1985). Although complete model validation is not possible, the revised model is consistent with experimental data on blood lead accumulation following dietary lead uptake among infants (Ziegler et al., 1978), and skeletal lead accumulation following controlled exposures in adults (Heard and Chamberlain, 1984). Given the available data, the model appears to provide the best estimates of PbB levels in children with continuous lead uptake over time.

Table 4-2 presents PbB levels predicted by the model for male children under different exposures, or lead uptake levels. The values in Table 4-2 include an adjustment for the propagation of maternal lead during pregnancy that persists throughout childhood. This adjustment was done using another kinetic model fit to blood lead data collected longtudinally in young children from birth to 27 months (Succop et al., 1987) and assuming a baseline maternal PbB level of 4 μg/dl (see Section C.6). The following equation adapted from Succup et al. was used:

$$dPbB_t = PbB_0 \, (e^{-\alpha t})$$

where: $dPbB_t$ = added blood lead increment propagated from maternal PbB at time t;

PbB_0 = baseline maternal PbB (4 μg/dl);

and the rate constant $\alpha = 0.072$.

The rate constant $\alpha = 0.072 \text{ mo}^{-1}$ corresponds to a mean residence time of $1/\alpha = 14$ months, or a half-life of $\ln(2)/\alpha = 10$ months. This almost certainly represents resorption of that fraction of prenatal lead burden sequestered in the neonatal skeletal tissues. The apparent mean residence time of lead in blood in infants is much shorter (see Section V.A.5).

No differences were found by the biokinetic model between the sexes except at older ages, and predicted PbB levels were highest among 2-3 year olds, consistent with results of NHANES II, the New York City screening program (Billick et al., 1979), and ongoing longitudinal study (McMichael et al., 1986). These results will be applied to estimates of the integrated lead uptake model to estimate children's PbB levels under alternative air lead levels. It is important to note that this equilibrium blood lead model is linear in total lead uptake at each age. This is a necessary consequence of

TABLE 4-2.

PREDICTED EQUILIBRATED BLOOD LEAD LEVELS (µg/dl) OVER TIME AMONG CHILDREN WITH CONSTANT LEAD UPTAKES[a]

	Lead Uptake (µg/day)							
Age	10	20	30	40	50	60	70	80
1	3.0	5.9	8.9	11.9	14.8	17.8	20.8	23.8
2	4.9	9.0	13.0	17.1	21.1	24.2	28.3	32.3
3	4.6	8.2	11.9	15.5	19.2	22.0	25.6	29.3
4	4.5	8.2	11.8	15.4	19.0	21.8	25.4	29.0
5	4.4	7.9	11.4	14.9	18.4	21.0	24.5	28.0
6	4.4	7.8	11.3	14.7	18.2	20.7	24.2	27.6
7	4.2	7.4	10.7	13.9	17.2	19.5	22.8	26.0
8	3.4	5.9	8.3	10.8	13.3	14.9	17.4	19.9
9	2.3	4.6	6.9	9.3	11.6	13.9	16.2	18.5
10	2.6	5.2	7.8	10.4	13.0	15.6	18.2	20.8

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[a] From Harley and Kneip (1985) after accounting for propagation of maternal blood lead during pregnancy (see text).

the linear pharmacokinetic model assumed by Harley and Kneip (CD, Appendix 11-A). The uptake/biokinetic model is thus logically consistent with aggregate and disaggregate linear total-uptake models discussed in the following sections.

Figure 4-1 compares the relationships between lead uptake and blood lead derived from the various studies on infants and adults. Despite the diverse nature of the populations, study designs, and methodologies, there is a fair degree of consistency in the relationships. Each study found that a linear function provided as good a fit, if not better, than other non-linear forms at the relatively low exposure levels investigated. Some experimental and epidemiological evidence suggests however, that the relationship between lead concentrations in tissues and cumulative lead intake is only approximately linear at low levels of intake, and that successive increments in intake or exposure result in progressively smaller contributions to blood lead concentrations (Azar et al., 1975; Moore, 1977; Gross, 1981; Sherlock et al., 1982). The curves drawn in Figure 4-1 for infants and adults do in fact include smaller slopes for lead uptake values above 20-40 μg/day. This curvilinear relationship may be due to increased renal clearance with higher blood lead (Gross, 1981), distributional non-linearities due to differences in lead binding sites in different tissues (Hammond et al., 1981; Marcus, 1985b; Manton, 1985), and/or to a sizeable pool of mobile lead in bone maintained more or less independently of uptake (Rabinowitz et al., 1977; Chamberlain, 1983). It appears however, that none of the mechanisms introduce significant non-linearities at blood lead levels below 30 μg/dl (Marcus, 1984, 1985a,c; Chamberlain, 1983) and that a linear mathematical model is valid for relatively low to moderate lead exposures (CD, p. 10-31, Appdx. 11A-2). As discussed above, at levels above 30-40 μg/dl, blood lead may be an inadequate index

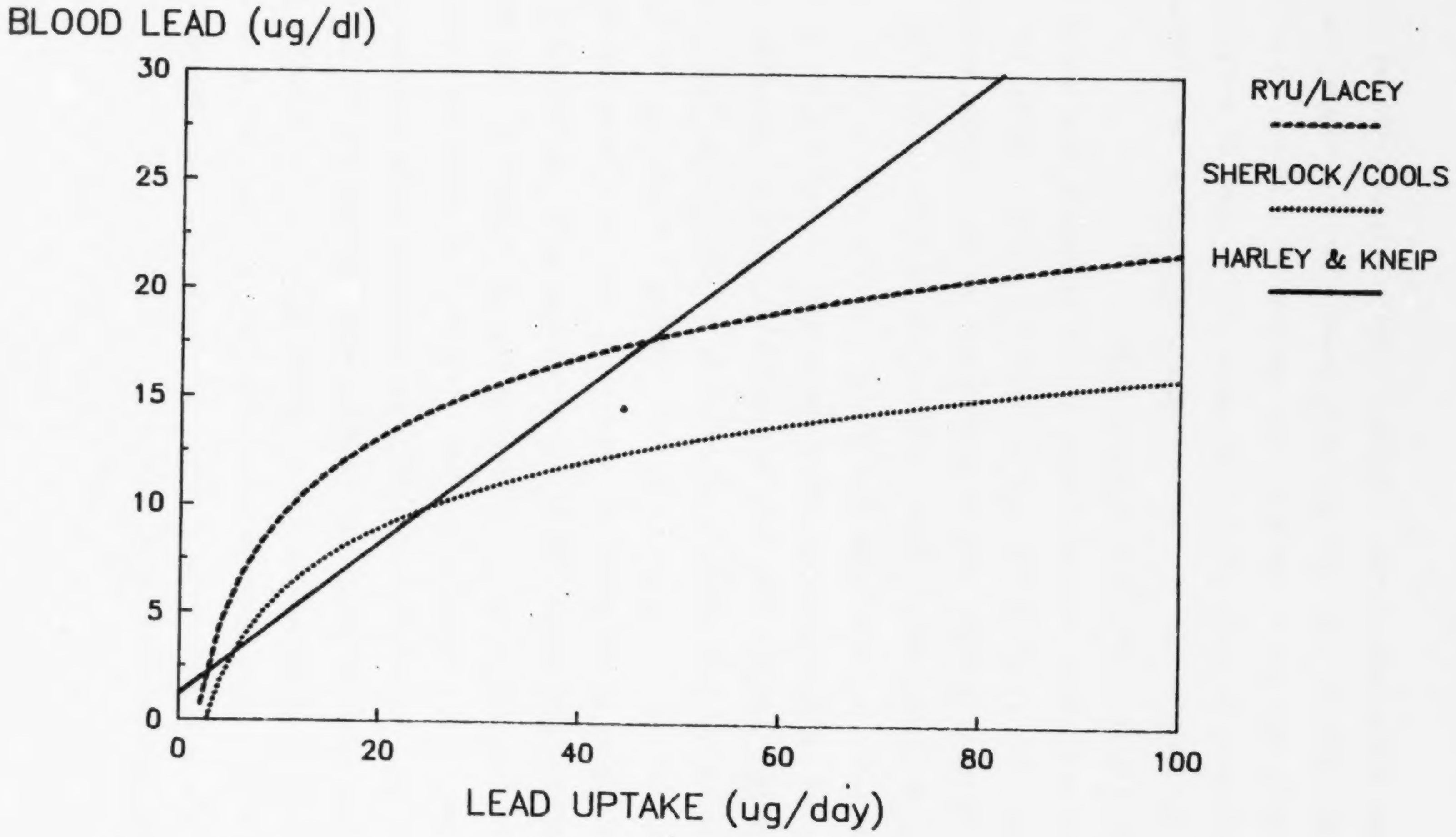

FIGURE 4-1. Summary of Relationships Between Daily Lead Uptake and Blood Lead for Different Age Groups, Derived from a) Ryu et al. (1983) and Lacey et al. (1985) Studies on Infants; b) Sherlock et al. (1982) and Cools et al. (1976) Studies on Adults; and c) Harley and Kneip (1985) Biokinetic Model with Estimates for 2-3 year old children illustrated.

for tissue lead burdens in many children (Piomelli et al., 1984) and linear models are likely to lose their predictive power. For this reason, the relationships depicted in Figure 4-1 are truncated at 30 μg/dl. To estimate PbB levels above 30 μg/dl, which is now above the PbB level of health-related concern for children, use of non-linear models discussed in the criteria document would be required (CD, Appendix 11.B).

The compartmental biokinetic model of lead metabolism developed by Mallon (1983) and Kneip et al. (1983), and revised for children by Harley and Kneip (1985), relied on a broad array of experimental and observational measurements of mammalian metabolism and growth patterns, and has been successfully validated using available human experimental and autopsy data. As is the case for any mathematical model, there are inherent limitations and associated uncertainties. Because this model is based on the most comprehensive data available and has been developed specifically to predict organ lead concentrations over time in young children with continuous lead uptake, it appears that the outputs of the Harley and Kneip (1985) biokinetic model are the most appropriate to predict PbB levels in children using the integrated lead uptake estimates presented in Section IV.A and Appendix A. PbB levels calculated from the conjunction of the integrated lead uptake and biokinetic models will be presented in the staff paper described in the Introduction (along with estimates using other modeling approaches that are discussed later) in order to estimate potential health impacts under alternative air lead levels. Methodologies for calculating children's and adult blood lead levels data under alternative standards using other data and other modeling approaches will be discussed in Section V.

V. STATISTICAL RELATIONSHIPS BETWEEN BLOOD LEAD AND AIRBORNE LEAD: USE IN DISAGGREGATE AND AGGREGATE MODELS

This section addresses two other methods of determining the contribution of air lead to total exposure and the potential health impacts associated with alternative air lead levels. The previous section examined the relationships between blood lead in children and estimated levels of lead uptake from the air, housedust, outdoor soil and dust, and diet. The two approaches presented here develop a direct relationship between air lead and blood lead from various experimental and community or epidemiological studies: 1) a disaggregate model in which total exposure to air lead is assessed by separately analyzing the relationship between blood lead and inhaled air lead, versus the associated changes in blood lead as a result of exposure to lead that has deposited onto soil, dust, food, and water; and 2) an aggregate model in which a single variable, air lead, serves as an index for lead exposure through other media affected by integrated atmospheric lead deposition.

Experimental studies include those in which adult volunteers have been exposed to controlled levels of laboratory generated lead aerosols. Because intake of air lead through the diet and dust was probably minimal for the adult subjects in these experiments, these studies underestimate the dose-response relationships that would be seen among children with changes in air lead (Angle et al., 1985). Epidemiological (i.e., community) studies provide correlations between air lead and blood lead in different populations of children and adults under varying conditions of lead, from current or previous atmospheric fallout and other sources, and yield dose-response relationships at more relevant exposure levels.

Because of the simultaneous presence of lead in multiple media with different time profiles, the assessment and use of these studies is not always direct. As in any attempt to model lead exposure, possible confounding variables that influence PbB levels but which are not related to air lead measurements (e.g., lead in paint, canned food and plumbing, socioeconomic status, parental care, housing and play conditions, calcium intake) cannot always be disentangled. Control for confounders can be achieved by comparing populations that differ only in their exposure to airborne-derived lead, although identifying such groups has been difficult. Another possibility is to perform a multivariate statistical analysis in which adjustments are made for some or all of the confounders before calculating the blood lead/air lead relationship. This requires information on the value of each confounder for each individual, which is also difficult to estimate. In the case of lead, several confounders tend to work in the same direction as air lead, e.g., elevated exposure and unfavorable social conditions often are both concentrated in central cities. Consequently, when statistical adjustment is incomplete, the relationship between air lead and blood lead will likely be inflated. Adjustment for a confounding variable may result in all of the shared variance between confounder and exposure variable being attributed to the confounder; this can inevitably lead to underestimation of the "true" impact of the exposure variable (Rutter, 1983). In characterizing good studies of air lead/blood lead relationships, several reviewers (e.g., Hammond et al., 1981; Brunekreef, 1984), as well as the CD, discuss key factors that should be considered. These include a well-defined study population, a good measure of individual exposure, measurement of blood lead with adequate quality control, a statistical analysis model that is biologically plausible and consistent with the data, and control or measurement of important covariates (CD, p. 11-63). The studies identified in the CD, and highlighted here, address these factors sufficiently to establish meaningful relationships.

A. Disaggregate Model

1. Air Lead/Blood Lead Relationships in Adults

Because the relationship between blood lead and environmental exposure is nonlinear across the range of potential exposure, but approximately linear at lower levels (CD, p. 11-65), the focus here is on studies of adults without excessive occupational or personal exposure. Longitudinal studies in which changes in blood lead were measured in adult volunteers exposed to controlled levels of laboratory generated aerosols, in some cases with isotopic lead tracer, are summarized in Table 11-40 of the CD. Data from the most relevant studies have been reanalyzed in the CD to yield blood lead air lead "slopes" (β), where β measures the change in blood lead expected for a unit change in air lead.

As noted in the CD, the blood lead inhalation slope estimates vary appreciably from one subject to another in the experimental studies, and from one study to another. The weighted slope and standard error estimates from the Griffin study (1.75 ± 0.35) were combined with those calculated similarly for the Rabinowitz study (2.14 ± 0.47), and the Kehoe study (1.25 ± 0.35), yielding a pooled weighted slope estimate of 1.64 ± 0.22 (CD, pp. 11-99 to 11-102). Excluding the subjects in the Kehoe study exposed to very high air lead levels (up to 36 μg/m^3), results in an average slope of approximately 1.9 μg/dl per μg/m^3.

Several deficiencies in the individual studies are discussed in the CD such as uncontrolled or unmeasured air lead exposures outside the chambers or difficulties in determining non-inhalation blood lead baseline levels (CD, p. 11-101). Furthermore, these experimental inhalation studies may underestimate the overall impact of airborne lead (via deposition and incorporation over time into soils, dusts, water, food chain). This is much less of a problem, however, in predicting changes in adult lead exposure in the future, compared to children. For example, although slightly elevated adult blood lead levels have been observed in areas of high lead contamination (Barltrop et al., 1975; Gallacher et al., 1984; Rabinowitz et al., 1985), adults do not ingest significant amounts of dirt and (unless under fasting conditions), normally do not absorb ingested lead at the same rate as children.

The cross-sectional relationship between blood lead and air lead in adults has been examined in several population studies. Azar et al. (1975) studied five groups of men (cab drivers and office workers) who carried personal air lead monitors and whose non-air lead exposures (water, smoking) were also measured over a 24-hour period. Several alternative geometric mean regressions have been calculated, including linear and non-linear models assuming different non-air contributions to blood lead, and estimations of the effect of endogenous lead stored in the skeleton using age as a surrogate measure of cumulative exposure. None of the fitted models are significantly different statistically (CD, p. 11-81) with a pooled slope estimate of 1.32 ± 0.38 µg/dl per µg/m^3 (CD, p. 11-105). Although the other population studies typically used less accurate measures of individual exposures (e.g., Tepper and Levin, 1975; Nordman, 1975; Johnson et al., 1975), the range of slope estimates (1.0-2.0) is comparable to the Azar et al. and experimental inhalation results (Snee, 1981; see CD, p. 11-98).

Although the studies differed in co-variables measured, populations, exposure conditions, etc., the most reliable and relevant studies consistently yield inhalation slope values typically in the range of 1.3-2.0 µg/dl per µg/m^3, with a weighted average slope of 1.4. The above slope estimates are derived from studies in which exposure to air lead was assessed over days, weeks, or months and are based on the assumption that an equilibrium level of blood lead is achieved within a few months after exposure begins. This is only approximately true, since some of the absorbed lead that accumulates in the skeleton deposits in the spongy trabecular bones (e.g., rib, vertebrae) where it may be resorbed into the blood stream. An additional factor therefore, should be allowed for possible re-entry of lead into the blood transferred from bone or other long-term storage (Chamberlain, 1983). Based on isotopic ratio and radiolabel tracer studies on adults, Chamberlain et al. (1978) estimated this factor to be 1.3. Similar experiments later indicated that about 20% (i.e., a factor of 1.2) of an adult male's blood lead is from bone lead resorption (Chamberlain, 1985). Re-entry into blood of stored lead is likely to be lower in children because of the rapid growth, high rate of turnover and smaller reservoir of lead in their skeletal systems. While no adjustment for bone resorption will be made for children's slopes, it appears reasonable to apply a factor of 1.3 to the blood lead/air lead inhalation slope of 1.4 derived above for adults, yielding a slope estimate of about 1.8 µg/dl per µg/m^3.

2. Estimating Future Adult Blood Lead Averages

Two adult populations are identified as especially sensitive to lead: middle-aged males, 40-59 years of age; and pregnant women, as exposure surrogates for fetuses (see CD, Sections 13.7.2 and 13.7.3). Older women may also be at increased risk due to mobilization of bone lead after menopause due to the processes of osteoporosis (Silbergeld and Schwartz, 1987).

Approximately 95% of adult body lead burden is sequestered in bone and this accumulation can maintain elevated blood lead levels years after high exposures have ended (CD, p. 10-23). In most cases, fetal exposure to lead can be expected to be dominated by maternal bone lead stores from past exposures which, in the U.S., were much higher than current levels. Accurately predicting future changes in fetal blood lead levels would require estimates of maternal lead burden and information on how bone lead stores would be transferred across the placenta. Although it is likely that there is extensive mobilization of lead, like calcium, during periods of physiological stress such as pregnancy, there are no biokinetic data to quantify this dynamic process. In the absence of such data, no attempt will be made to estimate fetal lead exposures associated with maternal PbB levels under alternative standards. Given the sensitivity of the fetus, however, potential risks associated with prenatal exposures will be of major emphasis in the overall lead NAAQS assessment.

Given a background or "baseline" blood lead estimate representing non-air lead adult exposures, and an average disaggregated blood lead: air lead relationship, the following simple equation can be used to predict blood lead means for adult male populations in our case studies:

$$\overline{x} = y_0 + 1.8A$$

where $\overline{x}$ = mean blood lead ($\mu g/dl$)

y_0 = non-air background blood lead ($\mu g/dl$), and

A = average air lead ($\mu g/m^3$)

Estimates of non-air "background" blood lead level contributions for different adult populations that will be used in the 1990-96 case studies are derived in Appendix C. Other issues involved in estimating fetal PbB levels are also discussed there.

3. Air Lead/Blood Lead Relationships in Children

Three population studies of children living near lead point sources in which covariates were controlled (e.g., age, sex, dust exposure) were extensively analyzed in the CD for the most useful and relevant estimates of inhalation slopes: Angle and McIntire, 1979 ($\beta = 1.92 \pm 0.60$); Roels et al., 1980($\beta = 2.46 \pm 0.58$); and Yankel et al., 1977/Walter et al., 1980 ($\beta = 1.53 \pm 0.064$). The median slope of the three studies is 1.97 (CD, p. 11-189); its application for use in the disaggregate model is discussed below.

4. Estimating Children's Blood Lead Levels Using Disaggregate Model

The relationship between blood lead and direct inhalation of airborne lead provides information useful for changes in air lead on a time scale of only several months (CD, p. 11-189). Over time, suspended lead is deposited and incorporated into soil, dust, and water, and enters the food chain. Since prior and current atmospheric fallout directly modify the daily burden of ingested lead, larger changes in blood lead would be predicted if the associated changes in the surface deposition of lead were accounted for, rather than simply inhaled air lead (Angle et al., 1984). To account for the simultaneous presence of lead in multiple environmental media, the CD has analyzed the separate mathematical relationships between blood lead and dietary, soil, and dust lead (described in detail in Chapter 11.4). Using representative values for lead concentrations in these media, these relationships were applied in a further analysis presented in the CD (CD, Table 13-6) as a way to estimate proportional inputs to total blood lead levels in U.S. children. A similar disaggregate model was developed by Angle and McIntire (1979) and Angle et al. (1984) in forming an integrated lead exposure function from measurements of lead in air, soil, and house dust and relating that to PbB levels of children living in various areas of Omaha.

The disaggregate model developed in the CD, intended to represent the then "current (i.e., 1983-84) exposure picture," is adapted here in Table 5-1 for different air lead levels and background levels of lead in food, water, dust, and soil, expected in 1990. The estimates are mean PbB levels for 2-year old children; comparable analyses for other ages of children can be done using the age-specific estimates of dietary lead intake presented in Appendix A. Calculations and assumptions used in deriving the estimates are summarized in footnotes to the table and are similar to those described in Table 13-6 of the CD. The following changes were made for the present analysis:

1) Estimated levels of dietary lead intake for children in 1990 were substituted for the estimates for 1983-84 used in the CD. The 1990 projections, described in Appendix A, are based on the Multiple Source Food Model developed in the CD and uses more recent data on the downward trends in food and water;

2) Soil and housedust lead concentrations associated with different air lead levels are estimated based on the long-term relationships derived from regression analyses of data collected from concurrent measurements of air, soil, and/or dust lead concentrations at about 45 lead point source locations (see Table A-4). These same relationships are being used to estimate surface soil and indoor dust lead levels in the uptake biokinetic model. In contrast, soil and dust lead concentrations under different air lead levels were estimated in Table 13-6 of the CD by interpolation of data from the Angle et al. (1984) study. This study is but one of many data sets included in the aforementioned regression analysis described in Appendix B.

TABLE 5-1. DISAGGREGATE MODEL OF CONTRIBUTIONS FROM VARIOUS MEDIA TO 1990 MEAN BLOOD LEAD LEVELS (μg/dl) OF U.S. CHILDREN (2 YEARS OF AGE): BACKGROUND LEVELS AND INCREMENTAL CONTRIBUTIONS FROM AIR[1]

	Air Lead (μg/m^3)					
Source	0.25	0.5	0.75	1.0	1.25	1.5
Background - nonair food and water[2]	1.5	1.5	1.5	1.5	1.5	1.5
Dust[3]	0.1	0.1	0.1	0.1	0.1	0.1
Subtotal	1.6	1.6	1.6	1.6	1.6	1.6
Background-Air Food and Water[4]	0.2	0.2	0.2	0.2	0.2	0.2
Ingested Dust (with Pb deposited from air)[5]	1.0	1.7	2.5	3.2	4.0	4.7
Inhaled air[6]	0.5	1.0	1.5	2.0	2.5	3.0
Total	3.3	4.5	5.8	7.2	8.3	9.5

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[1] Adapted from Table 13-6 of CD.

[2] Estimated dietary intake of non-air Pb in 1990 (from Cohen, 1988a,b; Appendix A) 9.2 μg/day x 0.16 μg/dl per μg/day (from Ryu et al., 1983 and Laxen et al., 1987 as re-analyzed by Marcus, 1989).

[3] From CD, Chapter 7, 1/10 dust not atmospheric. Using Angle et al. (1984) "low area" for soil and house dust levels and median regression coefficients from Stark et al. (1982): (1/10) x (97 μg/g x 0.0022) + (324 μg/g x 0.0018).

[4] As in 2 above, but using 1.2 μg/day for dietary intake of atmospheric lead. Derived for component of background Pb in food from past deposition from air onto soil and into other media leading into human food chain.

[5] Regression equations of Stark et al. (1982) used along with levels of soil dust and house dust predicted by regression analyses of collected point source environmental data, described in Appendix B. For example, the mean soil and house dust lead concentrations associated with 1.0 μg/m^3 in air are approximately 630 ppm and 1035 ppm, respectively.

The effect on blood lead would be:

(630 x 0.0022) + (1035 x 0.0018) = 3.2 μg/dl per μg/m^3.

[6] Using the median inhalation slope of 1.97 μg/dl per μg/m^3 from the CD, p. 11-189.

3) The soil and dust/blood lead regression coefficients used to compute soil and housedust contributions to blood lead are from the CD analysis of the Stark et al. (1982) study and are described in the CD as the most reasonable median estimates (pp. 11-151, 11-191). The coefficients used in Table 13-6 of the CD are from the Angle et al. (1984) study and represent upper bound values.

It must be emphasized that the blood lead estimates presented in Table 5-1 represent population mean PbB levels and are illustrative only. To assess health risks associated with such air lead exposures, it would be necessary to calculate PbB distributions around these mean levels, as discussed earlier in Section III.B.

The uncertainty in predicting mean blood lead using the disaggregate model can be estimated in principle since standard errors associated with the air Pb:PbB slopes are available (CD, p. 11-105). The differences among the slope estimates from different studies can be ascribed to many factors including differences in study populations, lead bioavailabilty, and to statistical artifacts, e.g., the attenuation of slopes attributable to environmental measurement uncertainties (Fuller, 1987). Differences in estimated standard errors of the regression coefficients reflect sample size, range of values of environmental variables in the sample, and control of other sources of variability. A composite slope could be estimated as a weighted average with weights inversely proportional to the variances for each of the slopes. Total variance would appropriately include both within-study and between-study variation and therefore all the variances and covariances of the parameter estimates would have to be calculated in a series of iterations. Although such analysis could be conducted, it was decided that the information about uncertainty of predicted mean blood lead added by this complicated procedure would not be significant, especially in view of the large uncertainties about the form of the dose-response model, the judgmental selection of key studies, and the contributions from non-air sources of lead.

Since completion of the 1986 CD, several findings have become available that supplement previous conclusions. Unpublished analysis by Johnson and Wijnberg (1988) of the 1983 study of young children living near the East Helena smelter (CDC, 1983) estimate slopes for both blood lead-soil lead and blood lead-dust lead of approximately 1.4 μg/dl per 1000 ppm. This analysis adopted modeling used by EPA in the CD to fit other similar studies and adjusted for numerous covariates including children's age, air lead, dust and/or soil lead depending on analysis, poor quality lead painted housing, smoking, and secondary occupational exposure.

As indicated in many of the studies, the dust lead slope is similar to the soil lead slope, suggesting that soil may contribute both directly and indirectly to blood. Such hypotheses regarding cause-effect responses of blood lead to earlier exposures have been explored in recent longitudinal studies on blood lead and developmental indicators in Cincinnati (Bornschein et al., 1985, 1986) and Boston (Rabinowitz et al., 1985; Bellinger et al., 1987) children. The Cincinnati data set was analyzed by structural equation methods (Bentler, 1980) for relationships between exterior surface scrapings, dust lead, and blood lead. Unfortunately, the results are expressed as a linear equation in logarithms of the environmental variables, and the standardized regression coefficients are not directly interpretable as overall slopes for soil or dust lead given the published information (Bornschein et al., 1986).

The analyses of the Boston data used a somewhat different technique--random effects models for longitudinal data (Ware, 1985). Unfortunately, soil lead data were not sampled longitudinally. There were repeat measurements for blood, dust, air and water lead. The 18-month blood lead regression model reported by Rabinowitz et al. (1985) used the logarithm of the yard soil lead measurement as a predictor, so that the reported slope is not directly comparable to those cited in the CD. However, the slopes at several concentrations are similar to those cited in the CD: 8 μg/dl per 1000 ppm soil lead when soil lead = 100 ppm, 1.6 μg/dl per 1000 ppm soil lead when soil lead = 500 ppm, and 0.8 μg/dl per 1000 ppm when soil lead = 1000 ppm. Subsequent analyses of this population using composite blood lead averages between 6 and 24 months (and a mean soil lead level of 700 ppm) yielded a slope of 0.9 μg/dl per 1000 ppm soil lead, varying from 0.6 to 1.6 according to the reported amount of mouthing by the children (Rabinowitz and Bellinger, 1988). Because the Boston study group did not generally live in crowded conditions, in homes with deteriorating leaded paint, or have nutritional deficiencies, their results may not be directly applicable to children at higher risk.

5. Plausibility of Disaggregated Slopes: Comparison with Uptake/Biokinetic Model

The disaggregated air, soil, and dust lead "slopes" used here are plausibly consistent with the uptake/biokinetic model parameters. To show this, we use the well-known relationship (e.g., Rabinowitz et al., 1976) between blood lead increments, dPbB, and lead intake increments, dPbI;

$$dPbB = dPbI * \text{(mean residence time in blood pool)} / \text{(volume of distribution in blood pool)}$$

For a two-year old child, the Harley-Kneip model predicts a mean residence time in the blood pool of about 8 days, i.e., a blood lead half life of 8 * 0.693 = 5.6 days, consistent with Duggan's (1983) estimate of 4-6 days. The blood lead pool is much larger than the blood lead volume, including accessible parts of the soft tissues, bone and bone marrow, and extracellular fluid. Rabinowitz et al. (1976) estimates that the volume of this pool in adult volunteers was 7.5 - 10.8 Kg (or approximately 70-100 dl), almost twice the adult blood volume. Thus, a factor of 2 is used to calculate the total volume of available blood lead distribution in the equation below. Since the blood volume in infants (about 12% of body weight) is proportionately greater than in adults (about 8% of body weight), we will assume the blood volume in 1-5 year old children is about 10% of body weight, adjusted for whole blood density of 1.06 kg/l. Thus, the volume of distribution in a 10 kg child (about 2 years old) is taken as about:

$$2 * 0.10 * 10\ kg/(1.06\ kg/l) = 2\ l = 20\ dl.$$

Assuming that the total amount of dirt consumed, C, is about 0.1 g/day, of which a fraction a = 0.25, approximately, is absorbed (see Appendix A.17), the leaded dirt is partitioned as some fraction p of soil and 1 - p of interior house dust. Thus:

$$dPbI = c * a * (p * PbS + (1 - p) * PbD)$$

Assuming p = 0.5 i.e. equal soil and dust access,

$$dPbB = (0.1\ g/d) * (0.25) * (0.5\ PbS + 0.5 * PbD) * 8d/20\ dl$$

$$= 0.005\ PbS + 0.005\ PbD$$

Thus the soil lead vs. blood lead and the dust lead vs. blood lead slopes are both predicted to be about 0.005 μg/dl per ppm.

A variety of factors may be responsible for the somewhat smaller empirical slope estimates, typically about 0.002 (see above). Even though the individual household PbS and PbD levels are available in some studies, these are not necessarily the exact exposure covariates, but only surrogate measures of dirt lead exposure. It is well known that exposure measurement "error" tends to flatten or attenuate regression slopes in dose-response studies (e.g., Pickles, 1982; Fuller, 1987; Brunekreef et al., 1987). Further, specification of the constants (e.g., $a < 0.25$ or $C < 100$ mg/d) could also reduce the predicted slopes. The agreement is nonetheless impressive.

The air lead slope is similarly calculated. From Table 4.1, the point source uptake at 1.0 $\mu g/m^3$ is 0.6 - 0.9 $\mu g/d$, thus

$$PbB = (0.6 - 0.9) * PbA * 8d/20\ dl$$

$$= (0.24 - 0.36)\ PbA$$

The estimated direct inhalation slope of 0.24 - 0.36 is much smaller than has been cited for empirical disaggregate studies. These discrepancies have been previously noted in Marcus (1988b) in an analysis of East Helena data. The differences between the uptake/biokinetic model apparent PbD slope and the disaggregate regression slope (0.00341 vs. 0.00151), apparent PbS slopes (0.00199 vs. 0.00102), and apparent PbA slopes (0.63 vs. 1.08) were in the same direction. It is possible that the empirical multiple regression studies have misallocated the variance in log(PbB), attributing too little effect to soil and dust ingestion. We cannot determine whether these are artifacts of the multiple regression model or of the uptake/biokinetic simulation model. Nevertheless, the slope values used in the disaggregate model are in general consistent with the uptake/biokinetic model.

B. Aggregate Model

1. Air Lead/Blood Lead Relationship

In the disaggregate modeling approach, "inhalation" slopes are derived from epidemiological studies by making adjustments for measured indirect air exposure variables (e.g., house dust lead). These inhalation slopes are then combined with separate slopes for other exposure media (e.g., dust, food, water) to arrive at an integrated lead exposure function. With the exception of Angle and McIntire (1979), the community studies from which inhalation slopes have been derived have not simultaneously measured lead in more than two or three media. Consequently, the integrated lead exposure function is necessarily based on data from multiple studies involving different populations, exposure conditions, measurement techniques, etc. An alternative method of calculating the effect of changes in air lead on children's blood lead is to analyze individual community studies in which reliable "unadjusted" blood lead/air lead relationships can be derived such that the impact of both direct (inhaled) and indirect (via dust, soil, etc.) contributions of air lead are combined, or aggregated, in one variable (i.e., air lead). This approach has the advantage over disaggregate modeling (using the adjusted "inhalation" slope) in that the total impact of atmospheric lead on children's exposure is better captured since since air lead levels, in general, are causally related to other important exposure variables such as hand and household dust lead (Brunekreef, 1984).

The population studies with identifiable air monitoring methods and reliable blood lead data are summarized in Table 11-36 of the CD. None of the studies included adult populations. Since adults do not typically ingest much deposited lead anyway, the aggregate modeling approach will be limited to young children. In Table 5-2, the calculated β values for each study are presented according to the blood lead levels, ages, and type of location of the investigated children. Different statistical analyses of these studies have resulted in a range of possible values for the blood

Table 5-2. BLOOD LEAD/AIR LEAD SLOPES[1] IN CHILDREN FOR DIFFERENT AGES AND EXPOSURES

Blood Lead (μg/dl)/ Age (years)	Type of Location (Study) Urban	Near Point Source(s)
<25/1-7	8.5[a,b] (Brunekreef et al., 1983)	3.6-4.0[a] (Zielhuis et al., 1979; Brunekreef et al., 1981)
<25/1-18	0.6 (Angle and 1.92 McIntire; 1979; 4.40 CD) 0.66[a] (Brunekreef, 1984 2.10[a] from Angle and McIntire, 1979)	-
<25/10-15	-	2.46 (CD from Roels et al. 1976) 5.3[a] (Roels et al. 1980) ≈ 5.9-9.8[a,b] (Brunekreef, 1984 from Roels et al., 1976, 1978, 1980)
>25/0-9	2.9 (Billick et al., 1979, 1980; Nathanson and Nudleman, 1980)	1.07-1.52 (Yankel et al., 1977; Walter et al., 1980; Snee, 1982b; CD) 2.4-3.3[a] (Brunekreef, 1984 from Yankel et al., 1977)
>25/1-18	-	2.6-3.7[a] (Landrigan et al. 1975; Morse et al., 1977) 4.6[a] (Brunekreef, 1984 from Roels et al., 1976, 1978, 1980)

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[1] Slopes are in units of μg lead/dl blood per μg lead/m^3 air.

[a] "Aggregate" slopes derived from group comparisons or multiple regression analyses that were unadjusted for soil or dust lead, thereby attempting to account for influence of deposited atmospheric lead through these exposure routes. Remaining slopes represent relationships between blood lead and air lead due solely to direct inhalation which have been calculated by adjusting for the influence of soil and/or dust lead.

[b] Use of low-volume particulate samplers likely underestimated air lead exposures, and overestimated the β value, especially near point source where large particles predominate.

lead/air lead relationship (β). Unadjusted (i.e., aggregate) relationships are presented in addition to adjusted (i.e., disaggregate) relationships derived from regression analyses, the latter which refer to the blood lead/air lead relationships due to direct inhalation exposure.

Relatively wide ranges of β values are observed for industrial and urban areas at low and high PbB levels and for all ages of children. The apparent trend towards smaller slopes with increasing PbB levels is consistent with findings within some studies (Angle and McIntire, 1979; Roels et al., 1980) and other observations of curvilinear blood lead/exposure relationships (Azar et al., 1975; Moore et al., 1977; Gross, 1981; Sherlock et al., 1982; Hammond et al., 1981).

As discussed, the CD determined that the disaggregate inhalation slopes derived from the studies by Angle and McIntire, Roels et al., and Yankel et al. are the most reliable given their overall quality. Aggregate analyses of these data, either by Brunekeef (1984) or the original authors (Roels et al., 1980) illustrate the significant decline in air:blood lead slope when adjustments are made for factors that covary with air lead (e.g., soil and house dust lead), and the dependence of these slopes on exposure levels. For example, Brunekreef (1984) segregated the Roels et al. data and compared groups with large differences in exposure (i.e., <1 km from smelter vs. urban/rural; β= 5.9) groups with small differences at high exposure levels (i.e., <1 km vs. 2.5 km from smelter; β= 4.6) and groups with small differences in exposures at low levels (i.e., 2.5 km from smelter/urban vs. urban/rural; β= 9.8). As in the Brunekreef et al. (1983) study, the use of low-volume samplers may have underestimated air lead exposures, and overestimated the β value, especially for those children near the smelter where large particles predominate. The slope may be underestimated for young children, however, since this study only sampled children older than 10 years of age.

It is important to note several additional studies and analyses that may provide equally relevant and useful information for purposes of estimating an aggregate blood lead/air lead slope. These studies (Brunekreef et al., 1983; Zielhuis et al. 1979; Brunekreef et al., 1981) used well-defined study populations (children younger than 10 yrs. old; PbB levels below 25 μg/dl), employed available quality control procedures for blood lead analysis, and measured or controlled for important covariates (see CD, Table 11-36). Several specific comments should be made regarding these studies:

1. The series of studies reported by Zielhuis et al. (1979) and Brunekreef et al. (1981) included many environmental measurements (lead in ambient and indoor air, lead in dustfall indoors and outdoors, soil, streetdust, floordust, tapwater, and dustiness of homes) and regression analyses to determine the impact of different variables on PbB levels (i.e., the above environmental indices as well as distance from the smelter, parental education, age of the child, mouthing activity, and cleanliness of the child). Venous blood lead samples were analyzed by standard techniques and although information on interlaboratory comparisons is not given in the original study, quality control participation is reported by one of the investigators in a subsequent review (Brunekreef, 1984). Air lead was measured at two sites 0.2 and 0.4 km from the smelter in 1976 and in 1977. The levels in Table 5-2 were taken from Brunekreef (1984) and represent those measured at 0.4 km from the smelter. Air lead was measured at 6 sites continuously for 2 months in 1978. Brunekreef (1984) estimates $\beta = 4.0$ for 1976 by assuming a difference of 2.0 $\mu g/m^3$ in average air lead exposure levels between the 2-3 year old children with the highest

and lowest PbB levels. After adjustment for parental education, the blood lead difference of about 8 μg/dl decreased to 7.2 μg/dl, resulting in a slightly lower β estimate (3.6). For 1977, a difference of 1.0 $\mu g/m^3$ was assumed for air lead exposure levels between smelter and control children aged 2-3 years who had PbB levels of 18.2 and 14.6 μg/dl, respectively, again resulting in a β of 3.6. In 1978, only children living between 0.4 and 1.0 km of the smelter were sampled and air levels did not correlate with blood lead within this population. Thus, no direct estimate of β can be derived, although soil lead and indoor and outdoor dust lead, and therefore, accumulated lead deposition, accounted for a significant fraction of the variance in PbB levels (Brunekreef et al., 1981).

2. The more recent study by Brunekreef et al. (1983) on Dutch city and suburban children measured venous blood (which was analyzed as part of the European Community laboratory quality control program) and many environmental and social variables and potential confounders (lead in drinking water, soil, street and playground dust, hand dust, indoor dust, mouthing behavior, dietary intakes, parental education and occupation, age of home, etc). The very high β value (8.5) derived by Brunekreef (1984) even after adjustment in a multiple regression analysis for six of the confounders, may be related to an underestimation of ambient air lead levels due to the fact that low volume British Smoke air monitors were employed, in contrast to the hi-vol samplers used in most other studies. The extent of inflation in the estimated slope as a result of any underestimated air lead level is uncertain. Brunekreef (1984) notes however that even if this bias is accounted for, the difference in urban and suburban air lead levels was probably not larger than 0.2 $\mu g/m^3$. The contrast in lead deposition between the areas was significant (i.e., 643 vs. 220 mg/m2/day),

indicating that ongoing lead pollution accounted for a good deal of the differences in PbB levels, although variations in historical emissions can not be entirely ruled out. The relatively low PbB levels in this study (≈ 8-13 μg/dl) could be another partial explanation of the high β estimates, given that other studies used to derive β included children with PbB levels on average closer to, and above, 30 μg/dl. At this point, the blood lead/lead intake relationship is estimated to level off; below 30 μg/dl, this relationship appears to be approximately linear (CD, p. 11-104).

The remaining slopes listed in Table 5-2 are based on data from children whose PbB levels, as some in Yankel et al. (1977), exceeded 25 μg/dl. As discussed in the CD, the relationship between lead uptake and PbB levels above about 30 μg/dl appears to be non-linear (CD, p. 10-31). Because PbB levels above 25-30 μg/dl exceed those where health-related concerns are triggered (see CD, Chapter 13), these slopes are less relevant to the present review.

In summary, the analyses using the aggregate approach assume the same source for most lead in air, soil, and housedust, and that adjustment of PbB levels for soil or dust lead to yield an inhalation slope, underestimates the "true" impact of atmospheric lead on PbB levels. A range of β values can be estimated from a) additional, and apparently relevant, studies not used to derive inhalation slopes in the CD (i.e., Zielhuis et al., 1979; Brunekreef et al., 1981, 1983) and b) aggregate analyses that include both direct (inhalation) and indirect (via soil, dust, etc.) air lead contributions in the key "inhalation slope" studies cited in the criteria document. Although far from conclusive, these studies and analyses suggest a range of possible blood lead/air lead aggregate slopes in the range of 2 to 10 for young, moderately exposed children with the most reliable slopes falling between 3 and 5 (CD, p. 11-104).

2. Predicting Children's Blood Lead Levels Using Aggregate Model

To predict PbB levels associated with alternative air lead levels using these aggregate slopes, it is necessary to estimate the contribution to total lead exposure from sources not affected by a proposed air quality standard, for example, lead-soldered food cans, lead in plumbing, and lead-based paint. It should be noted that by using measured lead concentrations in dust from a wide range of locations and conditions, the integrated uptake model discussed in the previous section implicitly includes average contributions to total exposure from lead-based paint, but excludes from the analysis high level exposures associated with deteriorated lead-based painted housing.

Several difficulties arise in attempting to explicitly estimate exposures from non-air sources of lead:

1) With the exception of non-air lead in diet, few studies provide detailed information on the relative contribution of various sources to children's PbB levels in the U.S. Estimates must be made by inference from earlier survey data; and

2) Because non-air contributions to PbB levels probably vary widely in space and time among children, a single estimate for the average case may result in a lead NAAQS that is not protective for all children. [This can be expected, for example, for children who regularly ingest lead-based paint.] Conversely, compliance with the air standard will provide extra protection in areas where lead from non-air sources is below the average.

In setting the 1978 lead NAAQS, the Agency estimated that non-air sources of lead contributed 12 µg/dl on average to children's PbB levels. Since then, significant reductions in PbB levels have occurred, attributable not only to declines in atmospheric lead emissions but to the gradual conversion by manufacturers to non-lead soldered cans, in some cases reduction in the number of old, lead-painted homes, cleaner working conditions, and by parents and public health agencies to minimize children's lead exposure. Further declines in lead exposure can be expected as these trends continue and lead exposure from drinking water drops, due to increased awareness as a result of widespread, mandated public notifications, the 1986 Safe Drinking Water Act banning the use of lead solder and pipes in new construction and plumbing repairs, and to reductions in water corrosivity by public water suppliers as compliance with the revised national water regulations for lead begins. The most recent, and well-conducted, nationwide survey of children's PbB levels was the Second National Health and Nutrition Evaluation Survey (NHANES II) of 1976 to 1980. Appendix C describes analyses that a) starts with the average PbB level for children from 1978, the midpoint of the NHANES II survey, and b) estimates a 1990 PbB average by adjusting the 1978 value to account for the important changes, summarized below, in lead exposure that have recently occurred and that can be expected to continue:

1. Use of lead in gasoline has declined by about 90 percent since 1978; this trend will continue up through 1990-92 as compliance with the lead in gasoline standard is completed and as the fleet of lead-burning cars shrinks. As observed during the NHANES II survey period (see CD, Section 11.3.6), the continued dramatic decline in gasoline lead emissions is predicted to parallel a major shift in PbB levels.

2. A significant reduction in dietary lead intake has occurred since the late 1970's, and this trend will continue as atmospheric lead emissions and deposition and the use of lead-soldered cans, continue to decline.

The derived range of mean PbB levels for children expected in 1990 (4.2 - 5.2 μg/dl) reflects changes that have occurred since 1978, and that are expected to continue, in gasoline lead emissions and deposition and canned food technology. Any other changes that are more difficult to quantify are also probably of lesser importance for the bulk of the population. For example, changes for children living in lead-painted housing has not been significant on a broad scale. This range of 1990 "baseline" average estimates represents the mean PbB levels that would be expected in 1990 in U.S. children not exposed to atmospheric lead from lead point sources directly or indirectly, based on the data and assumptions presented above.

Another method to estimate children's mean "non-air" PbB is to use available data on typical background levels of lead in food, water, dust, and soil ingested by U.S. children and the relationship between lead taken up through these media and children's PbB levels. Table 13-6 of the criteria document uses such data in calculating a mean PbB of 4.42 μg/dl to be expected at an air lead level of zero. Although this value was calculated based on 1983-84 FDA data on lead in food, it is interesting that it is within the range described above. This range can be used in the aggregate model approach to estimate ranges of mean blood lead levels under alternative air lead exposures in the following equation:

$$\text{mean PbB} = (4.2 - 5.2\ \mu g/dl) + (3 \text{ to } 5\ \mu g/dl \text{ per } \mu g/m^3)\ \text{Air Pb}$$

It must be emphasized that these estimates for non-air contributions to average PbB levels represent average values. Many children may be at risk for significantly higher lead exposures that cannot be prevented by atmospheric emission controls. In particular, these include children who live in deteriorated or recently resurfaced lead-painted housing who deliberately or inadvertently ingest paint dust through normal mouthing activities, children exposed to high drinking water lead levels from eroding lead pipes or solders in distribution systems, children of parents who work in lead-related industries and are exposed to lead dust that is subsequently carried home on clothing, or children living near lead smelters and other point sources where historical accumulations of lead are excessive. It is important that other regulatory agencies and public health programs, including other EPA components, responsible for minimizing children's lead exposures from non-air sources, or from historical accumulations of atmospheric lead deposition, maintain or increase, where necessary, their efforts.

VI. VALIDATION OF INTEGRATED LEAD UPTAKE/BIOKINETIC EXPOSURE MODEL

While all three modeling approaches identified in Section III are useful in assessing the relative protection afforded by different lead NAAQS, the aggregate and disaggregate blood lead-air lead models are intended to be "equilibrium" models. However, we know that there is a great deal of variation in past and present exposures to lead from various sources and pathways. Of the three, the uptake/biokinetic model can best estimate the changes over time of blood lead to changes in environmental lead in rapidly developing young children.

A general description of the model is provided in Section IV and Appendix A. Average daily uptake for young children is calculated under conditions specified in terms of ambient air lead levels, soil and dust lead levels that correspond to both historical and current atmospheric lead emissions, and dietary lead levels from both water and food. Additional exposures to paint lead can be added but are not in the present exercise given the high degree of variability, and the inadequate data base (see section II.F). Blood lead (PbB) levels in populations of young children, who are the most exposed and highly susceptible to lead, are estimated over time based on total daily lead uptake using a biokinetic compartmental model.

Parameters such as indoor air lead exposure, time spent indoors vs. outdoors, absorption rates through the lung or gastrointestinal tract, and amount of dirt that children typically ingest through hand-to-mouth activity, are estimated from available data in the literature, as summarized in Appendix A.

In this chapter, results of several validation exercises where predicted and observed blood leads were compared are presented. The most detailed analysis was performed with data gathered around a smelter in East Helena, Montana. Two types of validation efforts were undertaken with this data set: 1) in the first effort, the best data regarding such model parameters as observed air, soil, and dust lead exposure estimates were used; 2) in the second validation effort, predicted levels of air, soil and dust levels were used to estimate PbB levels. The latter work was undertaken to determine how well the model behaved when actual measurements of necessary input data are not available. This was necessary so that the reliability of the model could be assessed for policy analysis purposes when less than full information is available to use with the model. Also, presented in this section are validation exercises using observed blood lead estimates in locations other than East Helena. While less effort was undertaken in these exercises, they nonetheless provide additional information on the reliability of the model and provide insight regarding the sensitivity of the model results to uncertainties in the input data. Given the many uncertainties in the input data and the biological variability that cannot be incorporated, the results of the validation exercises presented below reveal that the lead uptake/biokinetic model performs quite well in predicting mean blood lead concentrations in children living near point sources of lead.

A. Validation Using 1983 East Helena Data

The data set considered first is based on a 1983 study in which the Montana Department of Health and Environmental Sciences (MDHES) and the Centers for Disease Control (CDC), in cooperation with EPA, measured blood lead levels in approximately 400 children ages 1-5 living around the ASARCO lead smelter in East Helena. The lead content of soil and dusts around and in individual homes was measured. Airborne lead was measured before and during the survey at 8 sites. Three study areas were designated according to their distances from the smelter: Area 1, within

1 mile of the smelter; Area 2, 1-2.25 miles from the smelter; Area 3, more than 5 miles from the smelter. The validation exercises included children from Areas 1 and 2 only.

In order to estimate 1983 PbB levels in the East Helena children so they could be matched against the measured PbB levels, exposure profiles were generated extending as far back as 1978, depending on the children's age in 1983 and their residential location. A five-year description of monthly ambient air lead concentrations throughout the study area was generated using the Industrial Source Complex-Long-Term (ISC-LT) dispersion model based on air lead concentrations, source sampling data of smelter emissions, and local meteorological data, and accounting for dry atmospheric deposition. Area and year-specific estimates of background lead contributions (i.e., mobile sources, re-entrained soil, local minor point sources) were also included to account for total ambient exposure. Only one site, about 1/4 mile from the smelter, provided sufficiently complete air quality data over the period 1978-1983 that could be considered representative of population exposure in the vicinity of the monitor although air lead data were available from monitors in ten other locations around East Helena. A total of 158 monthly average lead readings were available from these monitors between 1980 and 1983. These averages were compared with predicted air lead concentrations from the ISC-LT dispersion model. As would be expected with a dispersion model using limited meteorological data, the model predicted no more than 37% of the month-to-month variability in monitor readings. However, simple linear regression predicting monitored readings as a function of a constant and ISC predictions, indicated that reasonably accurate estimates of air quality were made with the ISC model: the constant was within the range of the estimated background concentrations and was statistically significant, and; the coefficient for the ISC prediction variable was not significantly different from 1.0.

Estimates of soil and dust lead concentrations were also obtained using two methodologies. In the first case, home-specific observed values for soil and dust lead were used. In the second case, soil and dust lead estimates were made using models developed by McLamb (1988) and Marcus (1988c) that describe soil and dust lead as a function of ambient lead. (For a description of these models, see Appendix B). The latter approach was deemed necessary to determine how well the model predicts when full information on model inputs is not available. (This is indeed the usual situation when the model is used to assess the impacts of alternative regulatory programs on mean blood leads of a population of children.)

Age-specific exposure parameters used as input to the uptake model are described in Appendix A. Estimates of dietary lead intake for different age groups between 1978 and 1983, listed in Table A-2, were calculated through application of the Multiple Source Food Model developed in Chapter 7 of the CD. The latter model uses year-specific FDA data on food lead content (i.e., market basket surveys) and age-specific data on dietary patterns throughout childhood and has since been validated using recent 1984-85 data (Flegel et al., 1988). The contribution of drinking water to dietary lead exposure was estimated for an average tap water lead concentration of 12 μg/l, which was the level reported for the State of Montana in 1983.

The criteria for validation need to be carefully defined. There is a great deal of individual variability in PbB levels, even with available estimates of lead concentrations in exposure pathways such as air, dust, and soil. The variability is attributable to individual biological variation, to unknown factors mediating exposure such as frequency of hand-mouth contact, and to other unattributed sources of lead. Even with good environmental data, the fraction of variance in the logarithm of blood lead that is accounted for by an optimal total uptake regression model (multiple R^2) rarely exceeds 30%. For the East Helena data, $R^2 = 0.28$ in the best fitting models (Johnson and Wijnberg, 1988). Thus, it does not seem fruitful to use the individual predicted PbB levels as a criterion for model validation. We used the more realistic goal of matching the geometric mean PbB for children in a large neighborhood that was moderately homogeneous with respect to exposure.

A more global validation involves comparing the cumulative distribution function of observed PbB with that of the individual PbB estimates from the uptake/biokinetic model. These comparisons from the different validation runs are summarized in Table 6-1.

The simulations were all run at two levels, one with all parameters set to the lower bounds for uptake parameters in Appendix A, and the second with all parameters set to the upper bound levels in Appendix A. The midpoint of the all-lower and all-upper bound PbB estimates, i.e., the simple mean was found to be the best predictor.

In model run A, only observed values for air, soil and dust input data were used. This data set was limited to the 28 observations near the smelter where an ambient monitor was within a few blocks of the homes. Model run B includes observations on 299 children. This expanded data set was obtained by using dispersion modeling techniques to estimate block specific ambient exposure estimates.[1] Comparisons between runs A and B illustrate that accurate PbB reductions result from using either observed

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[1] A similar validation exercise was conducted previously where average PbB levels predicted by the uptake/biokinetic model for different census tracts were compared to measured PbB levels in 1-5 year olds living near two secondary lead smelters and in a reference area, in Dallas. Results were useful in indicating that greater refinement in the spatial scale would be necessary in accurately modeling high exposure situations. For the East Helena analysis, and in subsequent case-study modeling, the highest degree of spatial refinement possible (i.e., city blocks or block groups) is used.

TABLE 6-1. COMPARISON OF INTEGRATED LEAD UPTAKE/BIOKINETIC MODEL PREDICTIONS TO 1983 MEASUREMENTS IN EAST HELENA

Model Run	Population[a]	Pb Air Quality	Dust/Soil Lead	Predicted vs. Measured Average Blood Lead[b] and GSD[c]
A	28 children living in area 1 around single air monitor with valid data	Measured at monitor approx. 1/4 mile from smelter	Measured in and outside individual homes	16.3 vs. 16.5 µg/dl* 1.42 vs. 1.49
B	89 Area 1 and 210 Area 2 Children	Contribution from smelter emission estimated by dispersion model (local meteorology data) local background Pb levels (e.g., auto emissions, fugitive dusts) estimated from 1982 source apportionment study	Measured in and outside individual homes	9.3 vs. 9.3 µg/dl* 1.61 vs. 1.59
C	89 Area 1 and 210 Area 2 Children	Same as for Run B except that dispersion modeling used meteorological data from a nearby airport	Estimated from generalized air: soil/dust Pb relationships from regression analyses of available data in literature	9.5 vs. 9.3 µg/dl* 1.67 vs. 1.59

Note: Dietary Pb exposure estimated for all model runs based on year-specific analyses of 1980's FDA food lead concentration data and food consumption data from Pennington (1983, 1986), and U.S.D.A. Nationwide Food Consumption Survey, 1977-78.

[a] Areas as defined in the text

[b] Geometric means

[c] Geometric Standard Deviation

*All differences statistically insignificant; t-tests of differences of means assuming lognormal blood lead distribution and non-equal variances.

or estimated ambient air data. Run C, like run B, has 299 observations. However, in the latter model run, observed measures of household-specific soil and dust lead are replaced with estimates obtained from equations that explain soil and dust lead levels as functions of ambient lead (see Appendix B). Also, while model run C uses dispersion model estimates of ambient concentrations, as does model run B, the source of meteorological data is different: Run B uses local meteorological data, and run C uses airport data. This difference allows runs B and C results to be compared to assess how sensitive blood lead estimates are to less location-specific meteorological data as well as to estimated soil and dust Pb concentrations.

Figures 6-1 and 6-2 compare the blood lead distributions predicted by Runs B and C, respectively, with the actual 1983 measurements of East Helena children. It is important to emphasize that the model, because of the available input data, is designed to predict mean population responses and not individual PbB levels. Individuals with greater than average responses in the upper tail of the distribution comprise the population of concern, however. As discussed in Section III.B., the response of the most affected individuals will be estimated in case-study analyses by calculating the lognormal distribution around a given mean PbB level using empirically-derived estimates of PbB variance. The degree of protection under a given exposure scenario will be characterized by the fraction of children with PbB below some level of concern. The results of the East Helena validation are shown for individual children in Figures 6-1 and 6-2, without the application of a GSD. The fraction, or percentage, protected is on the vertical axis and the potential level of concern is on the horizontal axis. As can be seen by these figures, and in scatter plots of individual predicted vs. observed PbB levels (not shown), there is significant individual variation at higher levels due to extra variability not included in the model. These figures reinforce the presumption

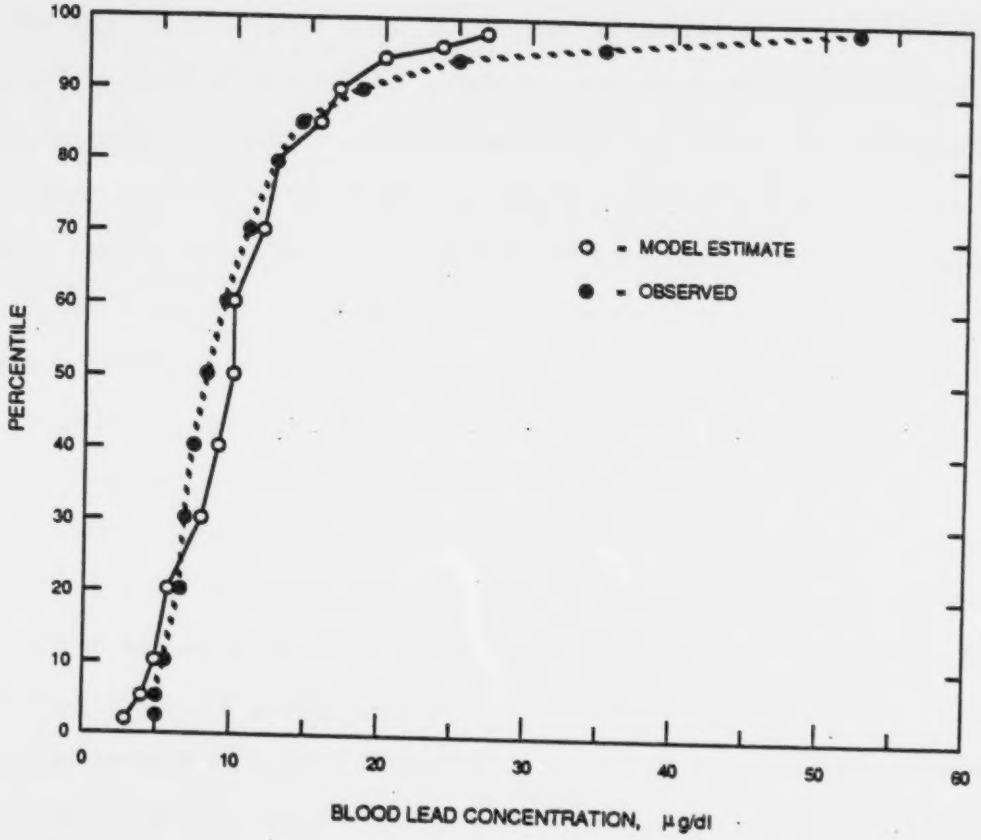

FIGURE 6-1. COMPARISON OF DISTRIBUTION OF MEASURED BLOOD LEAD LEVELS IN CHILDREN, 1-5 YEARS OF AGE, LIVING WITHIN 2.25 MILES OF E. HELENA LEAD SMELTER VS. LEVELS PREDICTED BY UPTAKE/BIOKINETIC MODEL. MEASURED SOIL AND DUST LEAD LEVELS WERE INCLUDED IN ESTIMATING UPTAKE LEVELS. (RUN B)

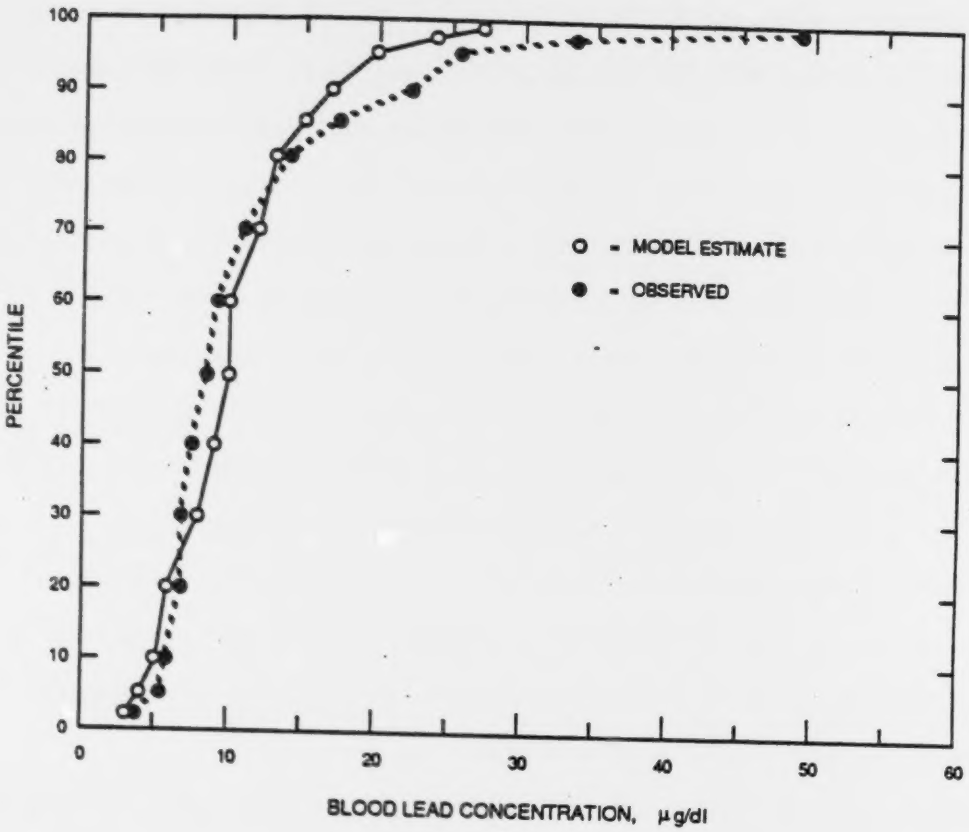

FIGURE 6-2. COMPARISON OF DISTRIBUTION OF MEASURED BLOOD LEAD LEVELS IN CHILDREN, 1-5 YEARS OF AGE, LIVING WITHIN 2.25 MILES OF E. HELENA LEAD SMELTER VS. LEVELS PREDICTED BY UPTAKE/BIOKINETIC MODEL. ESTIMATED SOIL AND DUST LEAD LEVELS FROM GENERALIZED RELATIONSHIPS WERE INCLUDED IN ESTIMATING UPTAKE LEVELS. (RUN C)

that the "importation" of empirically-derived PbB variance is necessary to fully capture variability. These figures also show that the model, as designed, gives a useful estimate of the mean response. Using the approach discussed previously of a) first estimating population mean PbB using the uptake/biokinetic model and b) then calculating the PbB distribution using an empirically-derived GSD (in this case 1.53; see Table 3-2), the modeled PbB distribution would be nearly identical to the observed distribution since the predicted mean is so close to the observed. Such comparisons are not illustrated here simply because the differences would be virtually indistinguishable.

In conclusion, generally satisfactory results were obtained for predicting geometric mean PbB levels. Model run A can be considered a "pure" validation run that uses the best input data available to predict mean blood leads which can be compared to mean observed blood leads. Model runs B and C were necessary validations since exact population locations, local meteorology, and soil and dust lead data will rarely be available for point source areas where the model will be used for policy analysis. Therefore, Run C can be considered a "generalized methodology" for "export" or application to other study areas.

Although the differences between observed and predicted mean PbB levels are not statistically significant, post hoc calibration of the model to remove any differences was considered (Marcus and Holtzman, 1988). Both linear and nonlinear models were examined and it was found that calibrated models (including age-dependent ones) improved predictive ability of the model only marginally. One alternative is to simply subtract the difference between observed and predicted PbB levels in the generalized methodology Run C (≈0.2 μg/dl) or use a multiplicative factor (the latter probably more appropriate given the lognormality of the PbB distribution). Such a calibration based on the results of a single, albeit large and detailed, validation may be inappropriate, especially considering the uncertainties inherent in such modeling exercises (e.g., a "positive" calibration may be needed in a different location). Given these uncertainties, no *post hoc* calibration of case study exposure estimates is currently being proposed although such an adjustment could easily be accommodated if decided upon.

Several sensitivity runs were made in order to "fine-tune" various parameters for which data are incomplete such as age-specific dirt consumption rates and gut absorption rates for ingested lead. For example, in estimating the rate by which ingested lead is absorbed, the criteria document identifies many factors that have to be considered, among them person's age, physiological status, medium for the lead (e.g., food vs. paint or "dirt"), coincident ingestion of other elements in food (e.g., calcium) which compete with lead for intestinal absorption sites, and the non-linear relationship between lead intake and blood lead (due for example to a saturable gut absorption pathway). Differences may also be attributable to physical or chemical differences in bioavailability of deposition from smelter emissions, or to the higher rate of intake in these areas compared to typical situations.

In fact, there are reasons to believe that such differences may exist in East Helena. The lead-bearing particles that are deposited near the smelter have a higher proportion of larger particles from fugitive emissions (e.g., erosion from tailings piles, loading operations) whereas the more distant deposits are predominantly smaller particles from stack emissions. Another factor is that parents of children who lived closer to the smelter (e.g., Area 1) may have been more sensitized to lead exposure and thus tended to control access to leaded soil and dust more than parents in

Area 2. There may also have been differences due to nutritional factors.) To test this, we ran the model with 20% gut absorption of dirt lead in Area 1 and 30% absorption in Area 2.

Two additional runs were made in which the same absorption factor, either 25% or 30%, was used for both areas. Area-specific results of the 20% (Area 1) / 30% (Area 2) model were not significantly different from the observed geometric means. Likewise, using a 25% absorption factor for both areas yielded no significant difference between predicted and observed geometric means for the overall study area. Results using the 30% rate cited in the CD based on indirect chemical and animal experiments were not as concordant. Because of the programming complexities that would be introduced if dual absorption rates were used in all other applications of the model, a 25% absorption rate is chosen for general use since it yielded adequate overall results.

As indicated in Section III, the model should be regarded as valid for estimating mean responses and it does appear that with the correct range of soil and dust lead inputs, the model adequately predicts the geometric mean of the observed distribution. Estimation of the higher percentiles of the PbB distribution requires application, to the geometric mean, of a GSD from, for example, NHANES II or exposure surveys around different point sources.

B. Application of Uptake/Biokinetic Model to Other Data Sets

There are other published data to test the uptake/biokinetic model at other U.S. locations and times. One data set was collected in Omaha, Nebraska, from 1971 to 1977 (Angle and McIntire, 1979; Angle et al., 1984). Another data set was collected throughout Silver Valley, Idaho, in 1974-1975 and is described in Yankel et al. (1977), and Walter et al. (1980) with additional information given in the CD.

1. Omaha Data

The Omaha study measured blood leads in 242 children ages 1-5 years and 832 children ages 6-18 years. The children were at 3 locations, denoted C (commercial area near a small battery plant), M (mixed residential-commercial), and S (suburban). Air lead, soil lead, and house dust lead measured in each area are incorporated into the model to estimate average daily lead uptake levels. These data are less satisfactory for testing the model than the East Helena data because the soil and dust lead measurements are not specific to each household where blood leads were taken. Furthermore, the study was not designed for controlled geographic comparisons given the significant demographic difference between areas in SES, housing, and racial composition. Age-specific dietary lead intakes were estimated based on 1970's data from the FDA and Jelinek (1982) as re-analyzed in the CD. In Table 6-2, the observed geometric mean blood lead levels for 1-5 year olds are compared to predicted mean PbB levels for 2-year olds in the suburban and mixed areas (blood lead data for 1-5 year olds were not reported for the commercial area). The model appears adequate for the suburban site, while average blood lead estimated from reported soil, dust, and air lead concentrations is significantly underpredicted for the "mixed" site.

There are several factors that may explain the contrast in PbB levels between the areas despite the apparent similarities in environmental lead levels, for example:

1) Air lead levels in the mixed area were apparently much higher than the suburban site in years prior to 1972 when soil, dust, and blood lead measurements were taken (e.g., 1.44 μg/m^3 in mixed vs. 0.73 μg/m^3 in suburban in 1970). Thus, the higher blood leads in the mixed area may

TABLE 6-2. CHILDREN'S BLOOD LEAD LEVELS MEASURED IN OMAHA, 1971-1977 VS. INTEGRATED UPTAKE/BIOKINETIC MODEL PREDICTIONS

	"Mixed" Site	Suburban Site
Average Air Lead (μg/m^3)	0.26	0.37
Average Soil Lead (ppm)	213	110
Average House Dust Lead (ppm)	653	567
Observed Mean PbB (μg/dl) 1-5 yr. olds	25.6	14.6
Predicted Mean* PbB (μg/dl) 2 yr. olds	20.2	15.1

Estimated Dietary Lead Uptake (μg/day) for both sites during early 1970's (see text)

0-1 yr. olds	16.3 - 20.6
1-2 yr. olds	20.2 - 25.6
2-3 yr. olds	16.6 - 22.1

*Predicted mean PbB represents midpoint of lower and upper bound means estimated by model.

reflect higher historical exposure; 2) The population in the mixed area was predominately black with a large percentage of substandard housing compared to the suburban area which was predominately white. Blood lead levels are highly dependent on demographic variables such as SES, race, housing conditions, parental care and nutrition, all of which were likely to have had systematic interactive influences in the Omaha study areas; 3) Other unmeasured variables may have differed between the areas such as drinking water levels (older homes in mixed area may have had lead plumbing), paint lead, frequency of canned foods in diets; 4) Possible measurement error related to the fact that most housedust samples were collected from vacuum cleaner bags. There may have been differences between areas in floor surfaces (carpet vs. wood) or frequency of vacuuming, for example.

2. Silver Valley Data

The Silver Valley study of 1974 and 1975 covered 860 children ages 1-9 years living near the lead smelter in Kellogg, Idaho and surrounding regions. PbB levels were quite high for children nearest the smelter. Household dustiness, soil lead, and estimated or observed air lead concentrations were obtained for 9 zones around the smelter and used to calculate daily lead uptake levels. As for the Omaha comparison, dietary lead intakes were calculated from 1970's data analyzed in the criteria document. The geometric mean values by zone are shown in Table 6-3, along with predicted mean PbB using the uptake/biokinetic model. Although comparisons in the three control areas (V-VII) are limited by the fact that air lead values were estimated there, results are generally adequate for the more distant areas with lower exposure levels. This is the intended range of applicability of the model. It is not surprising that blood leads are overpredicted in areas I and II located closest to the smelter. The Harley and Kneip compartmental model's presumption of linear kinetics in lead absorption,

TABLE 6-3. CHILDREN'S BLOOD LEAD LEVELS MEASURED IN SILVER VALLEY, IDAHO 1974-1975 VS. INTEGRATED UPTAKE/BIOKINETIC MODEL PREDICTIONS

Area	I	II	III	IV	V	VI	VII
Average Air Lead (µg/m^3)	16.8	14.2	6.6	3.0	0.7[a]	0.5[a]	0.5[a]
Average Soil Lead (ppm)	7,470	3,300	1,250	1,400	2,300	337	700
Average House Dust Lead (ppm)	11,700	10,300	2,400	3,300	3,400	1,800	3,900
Observed PbB (µg/dl) 2-yr. olds	72	51	36	35	35	25	35
Predicted Mean PbB[b] (µg/dl) 2-yr. olds	88.5	74.1	28.4	33.7	34.6	22.3	35.8

Estimated Dietary Lead Uptake (µg/day) for all sites during early 1970's (see text)

0-1 yr. olds	16.3 - 20.6
1-2 yr. olds	20.2 - 25.6
2-3 yr. olds	16.6 - 22.1

[a] Air lead concentrations in 3 control areas were estimated by ISC dispersion modeling.

[b] Predicted mean PbB represents midpoint of lower and upper bound means estimated by model.

excretion, and accumulation, while appropriate at relatively low to moderate exposures, is not applicable for such extraordinarily high exposure levels as observed in these two areas. Some adjustment for the non-linear patterns of lead uptake would be necessary in order to reliably model PbB levels in locales with such extremely high air, soil, and/or dust lead concentrations.

3. Other Data

Unpublished analyses by Phillips and Vornberg et al. (1986) for populations living near a large industrial lead source in Herculaneum, Missouri have similarly provided fairly good validation for the uptake/biokinetic model, especially when measured soil and dust lead values were available.

C. Conclusions

The above discussion illustrated several validation exercises that were undertaken to refine data inputs and to assess the reliability of the blood lead uptake/biokinetic model. The East Helena analysis was by far the most intensive effort. The results of the analysis indicated that the model, using the best available and location-specific input data, predicted overall means essentially identical to the observed blood leads. When using less location-specific input data (that is generally available for policy analyses), the model predicts mean PbB levels within 2 percent of observed. Other validation efforts, though undertaken with less effort, of data from Omaha and Silver Valley also support the hypothesis that the uptake/biokinetic model is reasonable for estimating blood leads when actual observations are not available. Other independent validation efforts have also provided general support for the efficacy of the model.

VII. CONCLUSIONS AND APPLICATIONS OF LEAD EXPOSURE MODELING

Because of the pervasiveness of lead in the environment, a large number and variety of exposure situations require some understanding not only in terms of present risks but for predicting hazards under future regulatory/abatement alternatives. More than perhaps any other environmental contaminant, a wealth of information has accumulated not only on lead toxicity but on lead exposure as well. This report has attempted to utilize the most reliable of that information to develop multi-media exposure methodologies to assess risks associated with alternative lead NAAQS and which can be applicable to other lead exposure scenarios. For example, blood lead levels in children exposed to different drinking water concentrations have been estimated in EPA's recent review of the lead in drinking water regulation by adopting both the disaggregate and the uptake/biokinetic modeling approaches. Similar applications may be appropriate for assessing the impacts of alternative soil lead contamination/abatement scenarios (Cohen, 1988c).

Each of the three approaches described here has both advantages and uncertainties. The aggregate model, for example, allows for a straightforward conversion of air lead concentration changes to the range of expected impacts on children's blood lead levels through direct and indirect sources. However, projecting in this approach what contribution non-air sources of lead will make to future PbB levels requires fairly good data on current exposures as well as trends in different exposure sources. Also, since soil lead changes relatively slowly in response to decreases in air lead, the change in blood lead calculated from the aggregate model may not be achieved for a long time. The disaggregate model, with rapid change in dust lead and slower change in soil lead, may be a more accurate predictor of near-term effects of air lead changes on blood lead levels. The uptake/biokinetic model, as with the other approaches, also must rely on incomplete data in some cases and because of its assumptions of linear absorption and biokinetics, is limited to predicting low-moderate level exposures (i.e., < 25-30 μg/dl). The model's mathematical assumptions and numerical parameters, however, combine plausible biological hypotheses, animal experimental data, and results of observational studies. Further, it allows explicit projections of future lead concentrations in various media and in turn can estimate impacts of these different changes on different age groups of children. It is this flexibility that makes the integrated uptake/biokinetic model adaptable for a wide range of predictive exposure assessments and why it was the focus of the validation exercises described in Section VI.

As discussed in Section I, the methodologies described in this report will be used to conduct case study exposure analyses around various lead point sources in the U.S. Blood lead distributions among young children and middle-aged men living near these sources in 1996 will be estimated under alternative lead NAAQS. Quantitative projections for pregnant women will not be made. Children's PbB levels will be estimated using the uptake/biokinetic and aggregate models; adult men will be modeled using the disaggregate approach. The range of GSDs (1.30-1.53) discussed in Section III.B will be used for children to calculate PbB distributions around the predicted means and results using the midpoint of 1.42 will be presented as best estimates; a GSD of 1.37 will be used for adults. In addition to mean PbB levels, percentages of the population above selected levels (e.g., 10, 15, 25 μg/dl) will be presented. Results of the case-study analyses will be included in the staff paper to help develop the range of standards for the Administrator to consider. Fetal exposures, although not quantified, will be a major consideration. Uptake/biokinetic model results for children will be used along with the adult men estimates in further quantitative analyses to estimate potential monetary benefits of alternative standards.

Results of the different validations indicate good concordance between observed and predicted average PbB levels in children living near lead point sources. The model may also be a useful tool in estimating PbB levels in children living with some other lead hazards, such as contaminated soils from historical deposition near major urban roadways or closed smelters or mines. Ongoing regulatory efforts by different components of EPA to control concentrations of lead in air, water and soil have created a significant need to model blood lead concentrations that delineates specific routes of lead exposure. At present, the uptake/biokinetic model provides the best method to achieve such specificity. Furthermore, such a model must be capable of simulating blood lead levels under future as well as historically known exposure regimes. (The validation efforts described in the previous section used historical data to test and improve the predictive ability of the model.) In fact, an earlier but similar version of the uptake/biokinetic model has been applied to assessments of Boston soil contamination (Beck and Tsai, 1987), exposures around the Herculaneum, Missouri smelter (Phillips and Vornberg et al., 1986) and historical contamination of soils near the Bunker Hill smelter in Kellogg, Idaho and as part of EPA's Integrated Environmental Management Project in their efforts to develop and apply methodologies to establish public health and environmental priorities.

Because the uptake/biokinetic model is linear, it cannot accurately predict excessive exposures (e.g., PbB levels above 30 μg/dl). Further development of the model to incorporate non-linear absorption and biokinetic patterns is underway. Other aspects of the model are still being assessed and validation tests will continue as new data are available. In particular, adjustments of dirt ingestion rate and dirt lead absorption may be desireable in calibrating the air, dust, and soil lead slopes relative to observed regression models or to available soil chemistry models (Cohen, 1988c). The empirical multiple regression methods used in the disaggregate models may also need refinement, e.g., by use of structural equation analyses that extend the statistical methods used by the Cincinnati Lead Program Project (Clark et al., 1987).

APPENDIX A. ESTIMATES OF LEAD UPTAKE

For each of the numbered lines in Table 4-1 in the integrated lead uptake/biokinetic model, the assumptions and estimates used in calculating average lead uptake for children from the various exposure pathways are discussed in the correspondingly numbered paragraphs below. Table 4-1 presents calculations for 2-year old children only; to estimate exposures throughout childhood, age-specific exposure parameters are necessary in several cases. These are also described below.

1. Outdoor air lead: In the case-study exposure analyses of alternative, future regulatory scenarios, ambient air lead concentrations will be assigned to populations living in different locations, or "receptor points." Locations will be defined as block group centroids - i.e., subdivision of census tracts. Air lead levels will be generated around individual lead point sources through use of the Industrial Source Complex-Long-Term (ISC-LT) dispersion model, based on site-specific operating parameters, emissions (both stack and fugitive), and meteorological data. Background lead concentrations from mobile sources, re-entrained soil, and local, minor point sources will also be included. The air lead concentrations specified in Table 4-1 do not represent exposure scenarios for alternative lead NAAQS but starting points to illustrate how daily lead uptake estimates are made.

2. Indoor air lead: The penetration of atmospheric lead into residential structures depends on the size of the lead particles, meteorological conditions, and the permeability of the windows, doors, and walls of the home. A range of indoor/outdoor ratios has been found (0.3-0.8) for different cities and structures (CD, Table 7-6). Near point sources where large airborne particles are more prevalent and infiltration into homes is low, the ratio appears to be closer to 0.3 (Cohen and Cohen, 1980).

3. Time spent outdoors: The amount of time spent between indoor and outdoor environments varies among young children depending on their stage of development (i.e., infant, toddler, pre-school), season, geographical location, and family behavior. While seasonal variations especially may be important in explaining summertime blood lead peaks among many children, yearly averages that smooth out such variations will be used for this modeling. There are several reasons: 1) There is little data to quantify seasonal differences in children's outdoor/indoor partitioning of time; 2) Regional differences confound seasonal variations in outdoor/indoor patterns, for example, a child in the South may spend more time outdoors in the fall and winter than in the summer, in contrast to a child living in a northern climate, 3) Although peak exposures are important, the model is being used to predict average exposures over several years. Such estimates would not be expected to be affected if seasonal differences were accounted for instead of integrating these differences to produce yearly averages. A range of 2-4 hours per day spent outdoors (and therefore 20-22 hours spent indoors) is considered a reasonable average for a 2-year old (CD, p. 7-43). The following age-specific estimated ranges for hours spent outdoors were derived from a literature review summarized in Pope (1985) using various studies (Hoffman et al., 1979; Rubinstein et al., 1972; Suter, 1979; Koontz and Robinson, 1982) and confirmed by informal surveying of parents:

age (years):	0-1	1-2	2-3	3-7
time spent outdoors (hrs/day):	1-2	1-3	2-4	2-5

4. Time weighted air lead concentrations for each level is estimated by: [(outdoor concentration x time spent outdoors) + (indoor concentration x time spent indoors)]   24 hours.

5. The volume of air breathed each day is dependent on age, body size, lung capacity, altitude, and activity of the child. For instance,

ventilatory volume can increase three-fold during strenuous exercise (Cotes, 1979). Phalen et al. (1985) determined average ventilation rates for males and females from birth through age 18 from graphical fits of published tabulated data (Altman and Dittmer, 1971, 1972). For example, a two-year old at "low activity" is estimated to have a minute ventilation rate of 2.75 liters/minute, which corresponds to an average daily rate of approximately 4 m^3/day while a three-year old is estimated to have a daily rate of 4.3 m^3/day. Other estimates that appear in the literature are 4.7 m^3/day (ICRP, 1975) and 4 to 6 m^3/day (Nutrition Foundation, 1982) for active one-year olds, and 4.7 m^3/day for a three-year old (Nutrition Foundation, 1982). These values combined with those determined by Phalen et al., and scaling factors based on body size are used to construct the following ranges for average age-specific daily ventilation rates.

Age (years):	0-1	1-2	2-3	3-4	4-5	5-6	6-7
Ventilation Rate (m^3/day):	2-3	3-5	4-5	4-5	5-7	5-7	6-8

6. The range of total lead intake by inhalation for each air lead level is computed by multiplying both the estimated upper and lower bound time-weighted average concentrations of air lead by upper and lower bound estimates of the volume of air respired per day.

7. Respiratory deposition and absorption: Only a portion of inhaled lead is deposited in the lungs and subsequently absorbed into the bloodstream. The deposition efficiency of lead particles depends primarily on their size and the physiology and rate of breathing of the individual. Available data on lead particle size distributions, particle deposition patterns in the lung, and respiratory absorption of lead particles were used to estimate deposition efficiencies of airborne lead particles in young children

(Cohen, 1987). A respiratory deposition/absorption rate of 25 to 45% is calculated for young children living in non-point source areas, while a rate of 42% is calculated for those living near point sources.

8. Total lead uptake from the air is the product of total intake and the lung deposition/absorption factor.

9. Average dietary lead consumption: Given the wide spatial and temporal distribution of food in this country, it appears reasonable to assume that even people living near industrial lead sources receive roughly typical lead levels from their diet. Any variability due to local contamination of garden crops or kitchens near point sources can be factored in separately. As noted in Section II.D., the Multiple Source Food Model developed in the CD has been validated and updated using the most recent food data from 1984 and 1985 (Flegel et al., 1988). Further declines in dietary lead intake are expected as a result of continuing reductions of lead in canned foods, gasoline emissions, and lead in drinking water. Cohen (1988a,b), uses available data on these downward trends and information from the Multiple Source Food Model on food consumption patterns, lead content of various foods, and source-specific contributions to project 1990-96 dietary lead intake estimates for different age groups of children. The age-specific estimates from Table 5 of Cohen (1988a,b) shown below as Table A-1 will be incorporated into the integrated uptake model to predict 1990-96 PbB levels among children. Similar dietary lead intake estimates were made for 1978-83 for purposes of validating the uptake/biokinetic model (See Section VI). These are shown in Table A-2.

10. Gut absorption of dietary lead: Only a portion of ingested lead is absorbed into the bloodstream from the gastrointestinal (GI) tract, or gut, and is dependent upon the composition of the diet and physiological

TABLE A-1. AGE-SPECIFIC ESTIMATES OF TOTAL DIETARY LEAD INTAKE (μg/day) FOR 1990-1996

Age (years)	"Metallic" (e.g. solder)	Atmospheric	Other	Total
<1	3.4	0.8	3.3	7.5
1	4.0	1.1	3.8	8.9
2	5.6	1.2	3.6	10.4
3	5.8	1.2	3.7	10.7
4	5.9	1.1	3.8	10.8
5	6.1	1.2	4.0	11.3
6	6.3	1.3	4.3	11.9

Source: Cohen (1988a,b)

TABLE A-2. TOTAL DIETARY LEAD INTAKE (μg/day) FOR 1978-1983 FOR FIVE AGE GROUPS OF CHILDREN[a]

Age (years)	1978	1979	1980	1981	1982	1983
1-2	45.8	41.2	31.4	28.8	26.0	19.3
2-3	52.9	48.0	36.9	33.8	30.6	24.1
3-4	52.7	47.8	36.9	33.7	30.6	23.0
4-5	52.7	47.8	36.9	33.8	30.7	22.0
5-6	55.6	50.3	38.7	35.5	32.2	23.2

[a] From Sledge (1986) -- calculated using year-specific FDA data on food lead content and Multiple Source Food Modeling methodology described in Chapter 7 of CD. Assumes average drinking water lead concentration at the tap of 12 μg/l. Used in validation exercises described in Section VI.

status of the individual. Based on measurements, mainly among infants, of lead in the diet and excreta, a gut absorption rate for ingested dietary lead between 42 and 53% has been calculated (Alexander et al., 1973; Ziegler et al., 1978). Corresponding rates between 7 and 15% have been estimated from adult studies (Kehoe, 1961; Chamberlain et al., 1978; Rabinowitz et al., 1980). Following the approach used by Harley and Kneip (1985), these rates were assumed for infants and adults and smoothed to yield values for intermediate ages, as follows:

Age:	0-1	1-2	2-3	3-4	4-5	5-6	6-7
Average GI Absorption Rate (%):	42-53	42-53	30-40	30-40	30-40	30-40	18-24

These rates do not reflect the wide degree of inter-subject variability observed in the studies nor other factors that influence absorption in children. (Such variability will be accounted for in calculating population distributions of PbB levels around estimated average PbB levels using empirically-derived geometric standard deviations). For example, absorption rates 2 to 4 times higher were observed in adults following fasting periods of 4 to 16 hours (Blake, 1976; Chamberlain et al., 1978, Heard and Chamberlain, 1982; Rabinowitz et al., 1980). Such increases can be expected among children who skip meals, a particular problem among lower income groups (Koh and Caples, 1977). Regular eating patterns do not ensure minimized dietary lead absorption, however. Based on several animal studies, clinical investigations, and epidemiological surveys, diets deficient in calcium, iron, phosphate, zinc, copper, vitamin D or protein, and excesses of dietary lipid or lactose, can be expected to increase the absorption and retention of lead (CD, Table 10-4). Data available from the 1976-1980 NHANES II indicate that as many as 22% of children aged 3-5 may have some form of iron deficiency (CD, p. 13-48).

This phenomenon may partially explain the enhanced neurotoxicity of lead in animals with nutritional deficits (Mahaffey and Michaelson, 1980). The importance of nutritional interactions with lead absorption and possibly toxicity is particularly significant for young children because of their large fluxes in relative nutrient status. Although nutritional deficiencies are more pronounced among lower income children, they exist in children of all socio-economic strata (CD, p. 10-41).

11. Daily dietary lead uptake is obtained by multiplying rows 10 and 9.

12. and 13. Dust/soil concentrations: As discussed in Section II, the accumulation of lead in street and household dusts and soils appears to be directly related to the volume of traffic, and inversely related to distance from neighborhood streets and roads, distance from lead based painted and brick houses and buildings, and distance from lead point sources. Predicting a relationship between different air lead levels and dust and soil lead levels over time would require the inclusion of many complex variables such as deposition rates, chemical and physical characteristics of the lead particles and soils, topographic and meteorological conditions, frequency of street washings and precipitation, background dust concentrations, and information on transport of dust and soil into homes and buildings. Given current data, there would be an extremely large amount of uncertainty surrounding any one of these variables for different locations. To predict outdoor and indoor dust concentrations under alternative air lead concentrations, reliance is placed on available studies that include measurements of both air levels and dust and/or surface soil concentrations (see Table A-3).

Because of historical accumulations of relatively large lead particles near primary and secondary lead smelters and other point sources, outdoor soil and dust lead concentrations, and consequently potential exposures, are significantly greater in these areas, regardless of ongoing emission

TABLE A-3. SUMMARY OF ENVIRONMENTAL LEAD MEASUREMENTS FROM VARIOUS POINT-SOURCE LOCATIONS

Average Air Lead ($\mu g/m^3$)	Average Surface Soil/Outdoor Dust Lead[a] ($\mu g/g$)	Average Housedust Lead ($\mu g/g$)	Location/Reference
			"Post-control"[b] (1973-1976) measurements in Omaha neighborhoods:
0.22	80	215	suburban
0.04	91	162	
0.32	591	280	mixed (battery plant in residential neighborhood)
0.46	260	470	commercial (Angle and McIntire, 1979; Angle, 1985)
0.30	114	-	"Post-control"[b] measurements (1976) in rural area, Brussels, and near lead smelter, Belgium (Roels et al., 1980)
0.45	112	-	
0.8	466	-	
3.67	2,560	-	
0.41	690 (street dust) 240 (soil)	1,239*	Near Arnheim secondary lead smelter, Holland (Brunkreef et al., 1981; Diemel et al., 1981)
0.82	924	713	Near Toronto secondary lead smelters and in city (Roberts et al., 1974)
3.01	2,416	1,550	
≈ 2.0[c]	1,214 (<1.6 km)*	-	Various distances from a lead-zinc smelter in British Columbia (Schmitt et al., 1979)
	698 (1.6 - 3.2 km)*	-	
	253 (>3.2 km)*	-	
1.6	-	1,825*	Near lead smelter in Yugoslavia (Prpic-Majic et al., 1984)
2.0	-	1,900*	
			Distance range (km) from zinc or copper smelters in:
0.13	35*	241*	(3.5 - 24.0)
0.20	243*	409*	(1.3 - 3.7) Bartlesville, OK;
0.30	829*	386*	(0.8 - 4.3)
0.31	821*	441*	(0.8 - 1.5)
0.14	75	235	(10.0 - 26.0)
0.18	115	164	(3.5 - 21.0) Anaconda, MT;
0.09	294	210	(2.0 - 11.0)
0.26	424	398	(2.0 - 3.5)
0.09	58	75	(3.4 - 68.0)
0.11	65	60	(1.0 - 6.4) Ajo, AZ;
0.19	77	65	(0.5 - 2.3)
0.26	95	116	(0.5 - 1.3)
0.36	532	263	(11.0 - 26.0)
0.56	117	201	(5.4 - 14.5) Palmerton, PA
0.13	326	198	(3.3 - 9.9)
0.28	331	438	(0.3 - 2.8)
			(Hartwell et al., 1983)

TABLE A-3. SUMMARY OF ENVIRONMENTAL LEAD MEASUREMENTS FROM VARIOUS POINT-SOURCE LOCATIONS (Continued)

Average Air Lead ($\mu g/m^3$)	Average Surface Soil/Outdoor Dust Lead[a] ($\mu g/g$)	Average Housedust Lead ($\mu g/g$)	Location/Reference
1.3	427	1,479	Near El Paso primary lead smelter "Post-control"[b]
2.7[d]	948[e]	8,623[e]	"Pre-control" (Landrigan et al., 1975; Morse et al., 1979)
0.5	337	1,800	Near Silver Valley (Kellogg), Idaho, primary lead smelter; Pre-control[b] Yankel el al., 1977; Idaho Dept. Health and Welfare, 1977)
0.5	700	3,900	
0.7	2,300	3,400	
3.0	1,400	3,300	
6.6	1,250*	2,400*	
14.2	3,300*	10,300*	
16.8	7,470*	11,700*	
0.3	200	501	Near E. Helena Montana primary lead smelter (CDC, 1983)
0.8	158	398	
1.9	307	891	
2.9	1,345	1,585	
3.6	1,549	2,284	
0.3	157	170	Near Herculaneum, Missouri, primary lead smelter (Phillips and Vornbery, 1986; Vornbery, 1987)
0.8	1,822	2,040	
0.3	70	850	
0.5	183	1,030	
0.8	2,239	1,210	
0.8	508	975	
2.2	2,558	1,610	
0.8	148	630	
1.1	827	1,600	
2.8	1,458	2,080	

*Data not used to develop air:soil:dust Pb relationships in Tables A-4 and A-5; See Text.

[a] Surface soil measurements represent top 0 - 2.5 cm.

[b] "Post-control" refers to the period of time following the application or increase in emission controls at the lead point source(s) involved in the particular study. Conversely, "pre-control" refers to the period of time before the application or increase in emission controls.

[c] Annual mean concentration reported as an approximate value

[d] Geometric annual mean of air lead levels measured within 5 km of smelter

[e] Geometric mean of dust and soil lead levels within 6.4 km of smelter

controls. This is the main reason why separate exposure estimates for individuals living near point sources under alternative air lead levels are required. Several data points collected from non-point source areas are excluded from Table A-3 and from subsequent analyses. These data include those collected near roadways in English and U.S. cities dominated, at that time, by automotive emissions, and in some cases, significantly impacted by old lead-painted homes (e.g., Barltrop et al., 1974; Lepow et al., 1975; Davies et al., 1987). Only data collected near lead point sources where emissions were comparable to current situations are used to develop outdoor soil/dust lead levels for point sources meeting different air lead concentrations (see Appendix B).

The techniques used in the sampling and pre-analytical treatment of dusts and soils influence the lead concentrations found. In order to make comparisons between different studies possible, analyses are limited to those studies that collected only the top 1-5 cm of soils (the most relevant depth for childhood exposure), collected dusts using surface wipes or vacuum pump, and employed careful analytical quality control. Although there are inevitable uncertainties in relying on a variety of studies, an attempt has been made to use the best available data to derive plausible relationships between air, soil, and dust lead concentrations.

Several of the studies besides the "non-point source" studies for which data are not used to develop relationships require comment: a) The Idaho smelter studied by Yankel et al. (1977) operated for several years with severely limited air pollution control capacity due to a baghouse fire in 1973. The very high soil and dust lead concentrations measured near the smelter would not be expected today with normally controlled emissions; b) Similarly, the pre-control measurements reported for the El Paso smelter are not relevant to operating facilities today. In addition, the air lead average for the pre-control period represented a different geographical area than represented by the soil and dust measurements; c) The high indoor dust lead levels measured by Brunekreef et al. (1981) and Diemel et al. (1981) near the Belgian smelter were probably due, in part, to extremely high lead content (up to 40% by weight) in peeling paint in old houses (built around 1900; the children in these houses were excluded from analysis of blood lead levels); d) The relatively high soil and dust lead levels in relation to concurrent air lead concentration measured near the Bartlesville smelter by Hartwell et al. (1983) was probably due to a drastic reduction of total suspended particulate emissions, from 1600 tons/year to 15 tons/year, just prior to the onset of the study; e) The air lead level reported by Schmitt et al. (1979) for the smelter area in British Columbia was only an approximation; f) the protocol for dust lead collection in the Yugoslovian study (Prpic-Majic et al.) could not be verified.

Another data set excluded from analysis is the 1983 air, soil and dust lead measurements taken around the Bunker Hill smelter complex in Kellogg, Idaho. These data were not used because the smelter ceased operation in 1981 and current soil and dust lead levels are still declining and thus do not represent steady state conditions.

With these exceptions, the studies used in Table A-3 appear to have sampled a broad spectrum of homes (e.g., both low and middle class, old and modern, with and without leaded paint--those with high paint lead levels were excluded) and neighborhoods that can be considered fairly representative of current U.S. conditions in point source areas. Emphasis is placed on data collected near operating primary and secondary lead smelters. Other lead sources were also used, such as zinc and copper smelters, that have dispersion patterns similar to the lead smelters, but at lower levels. The studies used for analysis had average air lead levels thought to be representative of each defined study area, or "neighborhood." The average soil and dust lead values are thought to characterize average exposures for children who spend time at several households or play areas within each "neighborhood." Although far from conclusive, the available data from point source studies suggest the long-term equilibrium relationships between air lead (PbA) and soil/dust lead concentrations shown in Table A-4. Appendix B contains a description of the derivation of these equations.

Short-term (several months) departures from long run equilibrium levels of soil and dust lead (from imposition of new alternative lead NAAQS, for example) are also estimated in Appendix B. The short-term relationships are shown in Table A-5. In general, a change in ambient lead will in the short term, only induce direct changes in dust lead. In the long term, the relationships in Table A-4 are appropriate. Unfortunately, insufficient longitudinal data exists to explicitly estimate the temporal relationship between ambient, soil, and dust lead.

The ranges in lines 12 and 13 are means using the long-term relationships in Table A-4.

14. It is estimated that a young child typically sleeps about 12 hours a day (Pope, 1986b), leaving approximately 12 waking hours in which he or she is capable of ingesting dirt. The time weighted concentration of lead in dust and soil that a child is exposed to is thus computed by: [(outdoor soil/dust lead concentration x time spent outdoors) + (indoor dust lead concentration x time spent indoors)]   12 hours.

15. Amount of dirt ingested: Hand to mouth activity (e.g., thumb sucking and finger licking) and immature dietary habits (i.e., the retrieval and subsequent consumption of food from dusty surfaces or soil), which are

Table A-4. LONG-TERM RELATIONSHIPS BETWEEN DUST, SOIL AND AMBIENT AIR LEAD*

Soil Lead = 53 + 510 (PbA)

Dust Lead = 60 + 844 (PbA)

Table A-5. ESTIMATING SHORT-TERM[1] RESPONSES IN EQUILIBRIUM SOIL AND DUST LEAD LEVELS FROM CHANGES IN AMBIENT AIR LEAD*

Change in Dust Lead = 638 (Change in PbA)

Change in Soil Lead = No Change

*Derivation described in Appendix B.

[1] Duration of several months or less.

normal behavorial characteristics of children up to five years of age (Lin-Fu, 1972), make soil and dust major sources of ingested lead for children (Charney et al., 1983). Good information on the amount of dirt a child eats in the normal course of a day is needed for an assessment of health risks associated with toxic materials in the environment. Based on a small number of direct measurements, Day et al. (1975) estimated that under average urban conditions (and after 30 minutes of normal playground activity), 5 to 50 mg of dirt transferred from a child's hands to a typical "sticky sweet", and estimated that a daily intake of 2-20 sweets would result in a dirt intake of 10-1000 mg. Lepow et al., (1974) measured a mean of 10 µg of dirt on the hands of 22 young children which would be ingested with each episode of hand-to-mouth activity. These authors estimated that a child puts its hand into its mouth ten times per day which would result in the ingestion of 100 mg (0.1 g) of dust and soil per day. An average estimate of 100 mg of dirt ingested daily by young children has been used in several documents (Drill et al., 1979; NAS, 1980; CD, Table 7-23) to represent a probable value for a "typical" child.

Recently, Binder et al. (1986) and Clausing et al. (1987) estimated children's intake of soil by measuring relatively non-absorbed elements with high concentrations in soil as tracers. Levels of aluminum, titanium, and either silicon or acid insoluble residue were determined in soil and children's feces and mass balance calculations were made to estimate soil ingestion. In both studies, soil ingestion estimates based on titanium were highly variable and in many cases, an order of magnitude greater than results using the other tracers. This suggests an additional source of titanium not accounted for in the studies (e.g., paint, toothpaste, talc, laboratory contamination) and that the titanium-based, outlying estimate should be given less weight.

For 59 children, ages 1 through 3 years, Binder et al. estimated mean daily soil ingestion based on aluminum and silicon to be 181 and 184 mg/day respectively; geometric means for the two elements were 128 and 130 mg/day. The mean estimate based on titanium was 1834 mg/day. Of the various factors and assumptions used in this study that could have contributed to an inaccurate estimate of soil ingestion (e.g., assumption that the tracer element absorption is negligible, and that there is introduction or loss of tracer element during processing), the assumption with the most significant potential impact is that dietary intake of the tracer elements is negligible. Binder et al. and Sedman (1987) cite evidence that these elements do in fact occur in the diet. The study design used by Clausing et al. provides an indirect control for such dietary intake; 18 nursery school children and 6 hospitalized children, without soil contact, ages 2-4, were sampled. The average of estimated soil ingestions based on all 3 tracers was 105 mg/day for the nursery school children and 49 mg/day for hospitalized children which was significantly different despite the small number of samples. If as the authors assumed, all of the "soil ingestion" by hospitalized children is background due to dietary and other non-soil sources, correction of the nursery school average for this background would result in an estimated average soil ingestion rate of 56 mg/day. If the same background is subtracted from the Binder et al. estimates to adjust for dietary sources of tracer elements, soil ingestion rates of approximately 80-135 mg/day result. For children in the 1-4 year old age group studied by Binder and Clausing, a range of soil ingestion between 80 and 135 mg/day will be assumed.

As noted by Binder, Clausing, Sedman, and colleagues, considerable uncertainty surround these estimates given for example, the small number of subjects studied, the lack of complete data on dietary intakes and gut absorption, and the uncertain representativeness of Dutch nursery school children to U.S. children. Nevertheless, the range of the estimates is narrow despite being derived from separate data sets and does suggest that they reasonably reflect average soil ingestion for this age group. Given that children eat a combination of soil and dust, and that the studies did not distinguish between those two media, the estimates would be inaccurate if concentrations of silicon and aluminum were different in house dust and soil. Until further data are available, it will be assumed that soil ingestion estimates from these studies reflect total "dirt" consumption rates.

To use the above estimates to determine dirt ingestion rates for other ages of children, data on age-related changes in blood lead (Yankel et al., 1977; Annest and Mahaffey, 1984; Billick, 1982; Quah et al., 1982) and the prevalence of mouthing behavior (Millican et al., 1962; Barltrop, 1966) were applied by Sedman (1987). Relative age-related changes in soil ingestion calculated by Sedman were applied to the range derived above for the 1-4 year old age-group to yield the following estimates:

Age (years)	0-1	1-2	2-3	3-4	4-5	5-6	6-7
Dirt Ingestion (mg/day)	0-85	80-135	80-135	80-135	70-100	60-90	55-85

The lower bound of zero for the youngest age group was chosen because contact with dirty surfaces is limited for the first 6 months or so until crawling begins. In informal discussions with clinicians and other researchers, including those who are involved in ongoing studies of soil ingestion in children, there was general agreement that these estimated ranges are reasonable averages given the available data and associated uncertainties. Further research on this issue that includes specific examination of tracer element metabolism and dietary intake, and house dust ingestion, has been stimulated by the Binder and Clausing studies. Preliminary results from this ongoing research that can hopefully clarify the estimates presented here are expected sometime in early 1990.

The above ranges representing average exposure estimates are derived from studies that did not include children with pica. Dirt ingestion rates for many children can be much higher. For example, among young children hospitalized for asymptomatic lead poisoning and exposed to three play environments which differed in their stimulus complexity (e.g., number and type of toys, availability of playmate), significantly more mouthing behavior occurred in the impoverished play setting (Madden et al., 1980). In particular, children with pica who display patterns of repetitive hand-to-mouth activity, or who deliberately ingest paint, plaster, paper and other non-food items including dirt, may be exposed to considerable amounts of lead compared to children who only inadvertantly ingest foreign substances. Pica occurs to some degree in a substantial percentage of young children. Data from NHANES II indicate that the percent of children with a history of pica is significantly higher for those 6 months through 3 years old (11.0%) than for those 4 through 5 years old (3.2%) and for children living in households with annual family incomes < $10,000 (11.9%) than for those in households with incomes > $10,000 (6.0%) (Mahaffey and Annest, 1985). Estimates for pica prevalence rates, based on more limited samples, range as high as approximately 10 to 30% in children 1 to 6 years old to 35-50% in those 1 to 3 years old (Millican et al., 1962; Barltrop, 1966). Children exhibiting pica for paint are of major concern because of the high levels of lead in some paints, with older painted surfaces containing lead in concentrations greater than 1-10 percent (10,000-100,000 ppm). [The allowable lead content of paint was set in 1978 by the Consumer Product Safety Commission at 0.06 percent lead (600 ppm)]. Pica for paint is believed to occur in episodes, possibly 2 to 3 times per week (NAS, 1972). It has been estimated that a child can consume somewhat greater than 1 gram within a 24 to 36 hour period, with cases reported of up to 20 grams within the same time period (Sachs, 1975) and that children with pica for paint may consume 1 to 3 grams per week (NAS, 1972). Although direct data on pica children are not yet available, a reasonable "worst case" estimate of 1 gram/day for soil ingestion in young children has been suggested (White, 1987).

Estimates of daily lead uptake under alternative air lead levels are presented in Table 4-1 only for children without pica. To calculate the contribution of lead in paint to the total lead exposure of children with pica, a worst-case estimate of 1 gram of paint chips consumed per day could be used. Future reductions in airborne lead emissions are not likely to significantly alter pica exposure to lead over the next few years and other regulatory alternatives to protect these children must be considered.

16. Lead intake from dust and soil is computed by multiplying the time weighted concentration of indoor and outdoor soil/dust concentrations by the estimated dirt ingestion rates listed above.

17. Gut absorption of dirt: Animal experiments indicate that lead of variable chemical forms in soil or dust is as available for absorption as food lead (Dacre and Ter Haar, 1977) and *in vitro* studies demonstrate that the acidity of the human stomach is adequate to extensively solubilize lead assimilated from soil and dust (Day et al., 1979; Harrison, 1979; Duggan and Williams, 1977). Based on these data and the fact that ingestion of such materials occurs other than at mealtimes, allowing for potentially enhanced absorption, the CD estimates that 30% of the lead ingested in dust and soil is absorbed in a child (CD, p. 10-10).

As discussed in the CD, the relationship between blood lead and lead intake is curvilinear across a broad range of blood lead values such that the relative change in blood lead becomes smaller as "baseline" blood lead or lead intake increases (CD, p. 10-53, section 11.4.3). In the lower range of exposures, there is no significant difference between curvilinear and linear relationships (CD, p. 11-99, p. 11-134). A number of biological factors may explain the curvilinear relationships such as an increasing fraction of blood lead as plasma lead when circulating lead rises, with greater movement of plasma lead to tissues, including bone, and increased lead excretion. Another plausible basis is gradual saturation of lead transport proteins in the GI tract as lead intake rises (CD, p. 10-13). In fact, improved fits of the uptake/ biokinetic model estimates to measured blood lead data from E. Helena resulted when GI absorption rates in children were differentiated based on proximity to the smelter which is a surrogate for lead intake level. The best fits resulted when a GI absorption rate of 0.3, equivalent to the average rate cited in the CD for children, was assumed for children living beyond a one-mile radius from the smelter, and a rate of 0.2 was assumed for children living within one mile. A close fit between observed and predicted blood lead levels also resulted when an average rate of 25% was used for the entire area (within 2.25 miles) of the smelter. It is of interest to note that a GI absorption factor of 0.17 has been estimated for paint chip ingestion in children (CD, p. 10-10). For children above 6 years old, a slightly lower value is needed to reflect the fact that GI absorption efficiency generally is much lower in adults. For children ages 6-7 years of age, a GI factor of 0.15 and 0.2 will be assumed depending, as discussed above for younger children, on their proximity to a lead point source.

An inverse relationship was found between particle size and gastrointestinal absorption of lead, especially in the range of 1-100 μm, such that a 6 μm dietary lead particle was absorbed five times as efficiently as a 197 μm particle (Barltrop and Meek, 1979). There is little information to assess whether the conversion in the physical form of atmospheric lead-containing particles once inside the complex geochemical matrix of soil or as dust would affect their bioavailability and, consequently, toxicity. Thus, atmospheric lead particles that have deposited will be considered to have absorption rates if ingested, independent of particle size.

18. Total lead uptake from dust and soil is obtained by multiplying rows 16 and 17.

19. Total lead uptake is the sum of rows 8, 11, and 18.

Paint Lead Exposure Estimates

No attempt has been made to separate lead paint hazards from generalized conditions in estimating soil and dust lead concentrations associated with different air lead levels. The major reasons are that 1) any changes in the lead NAAQS will have minimal effects on children exposed to dusts and soils contaminated by the flaking, peeling, weathering or "powdering" of lead-based paint and 2) data are not available to adequately estimate lead concentrations resulting from lead paint contamination given their high variability depending on many factors including housing age, extent of deterioration, layers of paint, family behaviors, and climate. For purposes of simply illustrating the magnitude of risks associated with lead-paint hazards in relation to generalized exposures around point sources or in other areas, one set of average estimates of lead levels in dusts and soils in and around homes with lead paint, presented in the CD, can be used. Based on studies of Hardy et al. (1971) and Ter Haar and Aronow (1974), the CD states that soil lead and household dust lead concentrations around and in lead-painted homes can be expected to average 2000 ppm (p. 7-62). If this soil and dust lead level is entered into the exposure profile for children in Table 4-1, assuming an air lead level of 0.5 $\mu g/m^3$, for example, and a gut absorption rate of 0.17 cited in the CD for paint chips (CD, p. 10-10), ranges of total lead uptake would increase from 11.5-26.7 µg/day to 31.2-51.1 µg/day. Even this large difference markedly underestimates leaded paint exposure for many children since children who eat flaking paint or gnaw lead-painted woodwork are not accounted for. Assuming pica behavior (1 gram/day ingested rate, see #15 above) in lead-painted housing conditions with soil and dust lead levels of 2000 ppm, the daily lead uptake estimates cited above increase to approximately 350 µg/day.

APPENDIX B. ESTIMATION OF THE RELATIONSHIPS BETWEEN SOIL, DUST AND AMBIENT LEAD

A key assumption in regulation of atmospheric lead concentrations (PbA) is that changes in PbA will be followed by corresponding changes in soil lead concentration (PbS) and interior house dust lead concentration (PbD) in the same vicinity. Since ingestion of soil and dust lead during normal hand-mouth activity is believed to be the major source of lead uptake in very young children (and worse when pica is present) it is important to make a quantitative estimate of the magnitude of the effects on these proximate causes of childhood lead exposure due to changes in PbA.

1. Mathematical Models for Soil and Dust Lead vs. Air Lead

The relationships depend on many factors, including time (t) and physical or chemical properties of the atmospheric lead particulates such as particle size and surface water solubility of the particulate lead matrix. We let x denote a generic set of particle properties. The fraction of airborne particles deposited on soil, per day, denoted DF(x,t), is in general a function of x and t. So is the fractional rate of removal of surface soil lead, denoted RF(x,t), by burial, runoff, resuspension, cleaning and other activities, and the influx rate (μg/g per day surface PbS from non-air sources) denoted IF(x,t). Finally, in order to calculate the Pb concentration in the atmospheric particulates, we also need the atmospheric particulate matter concentration in particle class x at time t, denoted PM(x,t). A formal model for combining these quantities is described next.

A plausible mathematical model for the rate of change of PbS(x,t) can be expressed by a differential equation

Rate of change of PbS = non-air influx + atmospheric deposition - soil removal or, symbolically,

$$dPbS(x,t)/dt = IF(x,t) + [PbA(x,t)/PM(x,t)]\ DF(x,t) - PbS(x,t)\ RF(x,t) \quad \text{(Equation 1.1)}$$

An equilibrium model may be obtained by setting dPbS/dt = 0 and solving the resulting equation (suppressing dependence on t),

$$PbS(x) = A_0(x) + A_1(x)\ PbA(x) \quad \text{(Equation 1.2)}$$

where

$$A_0(x) = IF(x) / RF(x) \quad \text{(Equation 1.3)}$$

$$A_1(x) = DF(x) / [RF(x)\ PM(x)] \quad \text{(Equation 1.4)}$$

Note that this implies that when there is more unleaded dust in the air (larger PM(x)), then the soil lead vs. air lead slope A_1 is lower. Likewise, a non-equilibrium model can be derived from equation 1.1. In the case when all of the parameters are independent of time, we have a relatively simple solution in terms of an exponential approach to the equilibrium concentration PbS(x) in Equation 1.2,

$$PbS(x,t) = PbS(x,0)\ \exp(-RF(x)t) + (1 - \exp(-RF(x)t)\ PbS(x) \quad \text{(Equation 1.5)}$$

There may be substantial variation in the apparent soil lead-air lead "slope" where PbS has not had sufficient time to reach near-equilibrium, say $t < 3/RF(x)$. Thus the soil lead removal time scale $1/RF(x)$ is a critical parameter.

Another consequence of this model is that particle properties (x) may be a factor in producing an apparently nonlinear relationship between total soil lead concentration PbS

$$PbS = \Sigma_x PbS(x)$$

and total air lead concentration PbA

$$PbA = \Sigma_x PbA(x).$$

From equation 1.2, the equilibrium relation is

$$PbS = \Sigma_x A_0(x) + \Sigma_x A_1(x)\ PbA(x) \qquad \text{(Equation 1.6)}$$

This produces an exactly linear relationship

$$PbS = a_0 + a_1\ PbA \qquad \text{(Equation 1.7)}$$

when

$$A_1(x) = DF(x) / [RF(x)\ PM(x)] = a_1 \qquad \text{(Equation 1.8)}$$

$$a_0 = \Sigma_x IF(x) / RF(x)$$

The assumption that $A_1(x)$ is a constant at each site, i.e., does not depend on particle properties x, is plausible but untested. Empirically, the relationship between PbS and PbA is nearly linear at low levels of PbA, but may be somewhat nonlinear at higher PbA (McLamb, 1988; Marcus 1988b) It is more plausible that PbS(x) is linear with the average concentration of lead in the particles, as in Equation 1.4 so that a linear relation between PbS and PbA is, at best, an approximation, unless all particles are in a single x class.

Similar but more complex calculations may be used to derive a linear PbD vs. PbA relationship.

2. Review of Data in The Literature

A number of data sets were examined that could be used to assess the rate at which soil lead (PbS) and/or house dust lead (PbD) concentrations change over time as a consequence of changes in air lead concentration (PbA).

Most of the reported studies are of marginal value. We will first describe the data sets we found less useful.

The Omaha studies by Angle et al. (1984) contain useful annual data, but the spatial and temporal averaging is at a very broad scale and much detail is lost. The lead dustfall data (PbDF, mg/cm^2) was available for most of the years in which PbA averages are given, but PbD and PbS were only measured at 37 locations in 1972. The data are shown in Table B-1. No relation between PbDF and PbA is evident.

The Roels et al. (1978, 1980) data also measured PbA for a number of years, but PbD only for 1976, so is not useful for showing PbD changes.

The El Paso smelter data (Morse et al., 1979; Landrigan et al., 1975; Landrigan and Baker, 1981) measured PbA, PbD, and PbS at several locations in 1972 and 1977. In 1972 many of the most heavily lead-burdened children lived in the Smeltertown neighborhood adjacent to the smelter. By 1977 almost all children had been evacuated to safer neighborhoods. In the intervening five years the mean annual air lead levels (PbA) had declined from 10.0 to 5.5 μg/m^3 at 0.4 km from the smelter. The dust samples in 1972 were collected in Smeltertown and other locations, with exceptionally high geometric mean PbD value of 22,191 ppm in the ring 0 to 1.6 km from the smelter. The 1977 mean PbD was only 1,479 ppm in this ring, and 1,461 ppm in the ring 0.8 to 1.6 km from the smelter. Likewise, the 1972 soil lead mean was 1,791 ppm in the 0 - 1.6 km ring, and 427 ppm in 1977.

The decline in El Paso PbS and PbD is proportionately much greater from 1972 to 1977 than the decline in PbA. Several factors may play a role here. The first is that the extensive publicity given to lead as a health hazard probably caused much more extensive individual attention to

TABLE B-1
ENVIRONMENTAL LEAD CONCENTRATIONS IN OMAHA, NEBRASKA (ANGLE ET AL., 1984)

Area	Year	PbA	PbDF	PbD	PbS
C	1970	1.66	-	-	-
	1972	0.37	-	479	262
	1973	0.46	26	-	-
	1974	0.04	17	-	-
	1976	0.13	5	-	-
	1977	0.78	34	-	-
M	1970	1.44	-	-	-
	1972	0.32	-	300	339
	1973	0.32	11	-	-
	1974	-	6	-	-
	1976	0.16	1	-	-
	1977	0.62	8	-	-
S	1970	0.73	-	-	-
	1972	0.26	-	211	81
	1973	0.22	7	-	-
	1974	0.04	3	-	-
	1976	-	-	-	-
	1977	-	-	-	-

household cleanliness and to control of areas in which children could be exposed to leaded soil (Landrigan, personal communication, April 5, 1988). Secondly, we do not know if the same locations were sampled in 1972 and 1977. Interior household dust collection would have been negligible in Smeltertown in 1977 as only three children lived there. Finally, the air lead levels relevant to the highest PbD in 1972 may have been much larger than 10.0 μg/m^3 in 1972. Landrigan et al. (1975) reported a mean PbA of 92 μg/m^3 at the air monitor closest to the smelter. A PbD deline from 22,191 to 1,479 ppm (15-fold) is better explained by a PbA decline from 92 to 5.5 μg/m^3 (17-fold). Access to unpublished data may clarify this.

The NEA source apportionment study (Cooper et al., 1981) obtained a number of low-vol, high-vol, and dichotomous sampler TSP samples at locations in the vicinity of the Kellogg, Idaho, smelter during 1980, when the smelter was still in operation. Samples were also obtained of "aerosolizable dust." These data are not directly comparable to PbS values. The samples of soil and road dust were sieved, aerosolized, and samples with a dichotomous sampler. This fine surface soil fraction probably is more relevant to the exterior dusts that adhere to a child's fingers and are eaten (Duggan and Inskip, 1985). Since a number of samples were obtained after the area had been covered by fine volcanic ash from the eruption of Mt. St. Helens on May 18, 1980, "Soil samples were collected after scraping away the overburden of ash which probably also removed the normal aerosolizable dust layer. Special care was exercised to ensure that the ash did not contaminate the underlying soil when sampled. The top one or two centimeters of soil were collected by scraping with a spatula..." (Copper et al., 1981, pp. 114-115). This study appears to be of marginal relevance for studying time trends.

3. Estimating Soil and Dust Lead Levels From Ambient Lead Levels

The assumption in most analyses of data around existing lead point sources is that the environmental lead emissions have been nearly constant for a sufficiently long time that lead levels in soil and dust are nearly in dynamic equilibrium. Thus, for some parameters a_0, a_1, b_0, b_1, b_2, c_0, c_1, we have the following linear relations for predicted geometric means depending on the level of available information:

Predicting PbS when PbA is available:

$$\text{G.M. PbS} = a_0 + a_1 \text{ PbA} \qquad \text{(Equation 3.1)}$$

Predicting PbD when both PbA and PbS are available:

$$\text{G.M. PbD} = b_0 + b_1 \text{ PbA} + b_2 \text{ PbS} \qquad \text{(Equation 3.2)}$$

Predicting PbD when only PbA is avaialble:

$$\text{G.M. PbD} = c_0 + c_1 \text{ PbA} \qquad \text{(Equation 3.3)}$$

Because these are predictions for geometric means and are fitted after a log transformation, we know that $c_1 \neq (b_1 + a_1 b_2)$. Note that $c_1 > b_1$ since it includes the indirect PbA --> PbS --> PbD pathway. The values shown in Table B-2 were obtained from two data sets. AGG refers to the 40 community averages from different lead point sources identified in Table A-3. Table B-3 summarizes the relationships derived from each of the locations. EH is based on a sample of households with young children obtained in 1983 in East Helena, Montana, by the Centers for Disease Control and the Montana Department of Health and Environmental Sciences (CDC, 1983).

These equations are not intended to be used recursively. If only PbA is available, then Equation 3.1 may be used to estimate PbS and Equation 3.3 may be used to estimate PbD directly, subsuming the intermediate PbA --> PbS --> PbD pathway. If PbS is in fact measured (such measurements being more often available than PbD), then Equation 3.2 can use the actual information in both PbA and PbS data without an intermediate estimate of PbS.

TABLE B-2
LINEAR MODEL PARAMETER ESTIMATES FOR AIR: SOIL AND DUST LEAD RELATIONSHIPS*

	Data Set	AGG	EH
Parameter	a_0	53.0	88.1
	a_1	510.0	206.0
	b_0	31.3	184.0
	b_1	638.0	267.0
	b_2	0.364	0.894
	c_0	60.0	220.0
	c_1	844.0	551.0

*From (Marcus, 1988c)

G.M. PbS = $a_0 + a_1$ PbA
G.M. PbD = $b_0 + b_1$ PbA + b_2 PbS
G.M. PbD = $c_0 + c_1$ PbA

TABLE B-3

AVERAGE DUST AND SOIL LEAD VS. AIR LEAD RELATIONSHIPS BY LOCATION[1]

		Dust		Soil	
Place	N	c_0	c_1	a_0	a_1
Ajo, AZ	4	46	195	41	204
Anaconda, MT	4	122	715	107	454
Bartlesville, OK*	4	139	974	-484	3989
Belgium	4	--	--	- 95	625
E. Helena, MT	5	263	418	65	268
Kellogg, ID	4	1488	613	278	407
Herculaneum, MO	10	220	934	-196	994
Omaha, NE	4	128	566	59	557
Palmerton, PA	3	144	566	225	648
Toronto, ON	2	400	382	365	681
Yugoslavia*	2	1525	188	--	--
All**	41	60	844	53	510

G.M. PbD = $c_0 + c_1$ PbA   G.M. PbS = $a_0 + a_1$ PbA

* Outliers: Dropped from analysis
**Includes Ajo, Anaconda, Belgium, E. Helena, El Paso (only one data point), Kellogg, Missouri, Omaha, Palmerton, Toronto

[1] Data from studies listed in Table A-3.

The data on time scales for soil and dust lead changes is inconclusive. Our opinion is that lead in undisturbed soil matrix persists for an extremely long time, but that soil lead concentrations in disturbed (especially urban) environments will change, on average, over periods of a few years to reflect changes in surface deposition. Interior dust lead concentrations will likely change over a period of weeks to months in response to air lead changes, depending on interior-exterior access and interior recirculation or removal of dust. Thus the following strategy is recommended.

The estimation of changes in PbS and PbD from a change in PbA (brought about from a hypothetical change in the Pb NAAQS) can be estimated under two situations--short-term and long-term. In the long-term, equations to use in predicting equilibrium levels of PbS and PbD following a change in PbA are illustrated by equations 3-1 and 3-3 above. Each predicts soil or dust lead dependent only on air lead, which will generally be the only information available in applied policy analyses. When available, PbS in combination with PbA should prove far more predictive of PbD than PbA alone, since the indirect pathway from PbA to PbD through PbS is probably a more important dust lead source (Rabinowitz et al., 1985).

The short-term changes in PbS and PbA, however, require further consideration. In time periods measured in terms of several months, we would expect to see little or no change in PbS from a change in PbA. Therefore, PbS would remain constant in the short-term. In contrast, dust lead, being a soiling phemonena, would be expected to change in the short-term as a result of changing ambient lead concentrations. There are, however, two components comprising the level of PbD. There is direct deposition from the atmosphere and secondary deposition caused by such factors as reentrainment from the soil. Therefore, the coefficient, b_1, in Equation 3.2 must be used to predict short-term changes in PbD (from equilibrium levels) from a change in PbA.

The model is intended to be used at many sites. Of the two data sets that were analyzed, the aggregate data set is preferable because it includes site-to-site differences in soil and dust slopes. Slopes from any particular location such as East Helena reflect local climate, soil type, accessibility of lead soil and air to the interior of the house, and housecleaning practices related to awareness of lead hazards. Thus, a model based on the EH data set may not be as transportable to other sites and the relationships expressed in Appendix A are based on the AGG analyses.

4. Conclusions

We have shown that the relation between soil lead, dust lead, and air lead is expected to vary over time, even under near-equilibrium conditions. The various slopes can be expected to depend on site-specific properties such as the deposition rate, removal rate, total airborne suspended particulate concentration, and non-air lead influx rate for lead-contaminated surface soils. There are almost no data that can be used to provide estimates of critical kinetic parameters such as soil or dust lead removal or turnover rates.

We thus suggest calculating the expected changes in PbS and PbD from equilibrium relationships. In the short term (a few months) we expect little or no change in surface soil Pb, but almost complete equilibration of the PbA (only) component of PbD and no change in the PbS component of PbD. In the long-term (e.g., ten years after a PbA change) we can use the fully equilibrated PbD vs. PbA slope that includes the indirect PbA --> PbS --> PbD pathway. The data from the aggregation of communities will be used since it will be more representative of the majority of point source locations.

APPENDIX C. ESTIMATING 1990 BASELINE BLOOD LEAD AVERAGES

1. Introduction

In estimating PbB levels expected under alternative lead NAAQS, both the disaggregate model for adults and the aggregate model for young children require estimates of the contributions to blood lead from sources that will be unaffected by changes in atmospheric lead related to change in the lead NAAQS. These sources include, for example, solder in canned foods and plumbing and historical deposition of gasoline lead. Blood lead contributions from non-air sources of lead are explicitly accounted for in the disaggregate model for children, and in the integrated uptake/biokinetic model. This section describes methodology for, and results of, estimating average blood lead contributions from non-air sources, or "baseline" blood lead levels for children and middle-aged male populations living in 1990. Estimates for women of child-bearing age are presented for 1) comparative purposes; 2) they could be used to estimate changes in fetal lead levels if biokinetic data during pregnancy becomes available; and 3) to adjust children's PbB estimates for recent reductions in maternal exposures. 1990 is the starting year for the various exposure analyses that will be conducted as part of the lead NAAQS review.

2. Approach

Ideally, we would know current PbB levels in pregnant women, middle-aged men, and young children living near point sources. Known blood lead distributions could then be adjusted to account for the continuing downward trends in food, water, and air lead concentrations, described in Section II, in order to estimate baseline 1990 blood lead distributions. Because there have been no recent systematic surveys of PbB levels in the U.S., baseline blood lead estimates will have to be based on earlier data.

The most recent, and well-conducted, nationwide survey of PbB levels was the Second National Health and Nutrition Evaluation Survey (NHANES II) of 1976 to 1980. Since then, lead concentrations in different media have dropped substantially and have apparently, and predictably, been accompanied by significant reductions in lead exposure. The approach taken here will be: 1) start with average PbB levels from 1978, the midpoint of the NHANES II survey; and 2) estimate 1990 blood lead averages by adjusting the 1978 values for changes in gasoline emissions, and food and water lead concentrations, that have recently occurred and that can be expected to continue. The issue of fetal lead exposure will be addressed separately.

3. 1978 Blood Lead Data

Table C-1 lists mean PbB levels for selected population subgroups measured as part of NHANES II in 1978, based on data provided to OAQPS by Joel Schwartz of EPA's Office of Policy, Planning and Evaluation. These levels will serve as starting points for adjustments to account for the exposure sources in flux, described above.

TABLE C-1. SELECTED 1978 BLOOD LEAD LEVELS FROM NHANES II

Population (Age)	Geometric Mean Blood Lead Level (μg/dl)
Children (0.5 - 5 years)	14.9
Females (15-44 years)	10.8
White Males (40-59 years)	15.4
Black Males (40-59 years)	17.7

Black middle-aged men have not been identified in the CD as a separate sensitive group. White middle-aged men have been the focus of the two key population studies on lead-related blood pressure effects (British Regional Heart Study and U.S. NHANES II) in order to avoid the effects of confounding variables and because of less extensive data for non-whites. Given their high risk of cardiovascular disease, it is likely that a similar, but possibly less apparent, blood lead/blood pressure relationship exists in black males and therefore, blood lead changes for this group will also be estimated.

4. Adjustment for Gasoline Lead Phasedown

The strong correlation between gasoline lead consumption declines and the downward trend in NHANES II PbB levels between 1976 and 1980 has been described in the 1985 Regulatory Impact Analysis for the final rule on lead in gasoline (EPA, 1985) and the CD (Section 11.3.6). Similar results were found with U.S. childhood lead-screening data (Schwartz et al., 1984) and in an Italian isotope experiment where fairly rapid changes in gasoline lead produced large changes in adult and children's blood lead (Facchetti, 1985; CD, p. 11-87).

Gasoline lead accounted for an estimated 60 percent of the lead in the average American's blood in the second half of the 1970's, and explained short-term seasonal increases in PbB levels from winter to summer as well as the long-term drop. Further, the accelerated rate of decline in gasoline lead after 1978 was paralleled by an accelerated decline in PbB levels. Additional support for a causal association between gasoline lead and blood lead is seen in a) the blood lead trend, which was consistent throughout the entire distribution, and not only through truncation of the high blood lead levels (CD, p. 11-185); b) one-month lagged gasoline lead sales being the most significant predictor of blood lead, which matches the one month half-life of lead in blood (EPA, 1985; p. 3-24) and is consistent with results of Rabinowitz and Needleman (1982, 1983) who found that monthly changes in gasoline lead exposure were the probable cause of trends in umbilical cord PbB levels in Boston births (CD, p. 11-39); and c) the gasoline/blood lead relationship being stable across demographic and geographic boundaries and between the first and second halves of the NHANES II survey, when gasoline lead levels were roughly 50% lower.

Regression coefficients estimated from the NHANES II analysis after controlling for age, race, sex, region of the country, season, income, degree of urbanization, and accounting for laboratory error, changes in dietary lead (including canned food) and lead-painted housing and other time-trends, have been used to predict changes in blood lead due to the gasoline lead phasedown (Annest et al., 1983; Schwartz et al., 1984). The phasedown will have continuing effects at least up to 1990-92 and earlier projections regarding blood lead changes can be extended using these regression coefficients. Joel Schwartz of EPA's Office of Policy Analysis provided the coefficients to OAQPS (personal communication to Jeff Cohen, May 9, 1988), derived from regressing natural-log blood lead values on gasoline values since the data best fitted a log-normal distribution. Similar analysis by the Centers for Disease Control and the National Center for Health Statistics has been described by Annest et al. (1983).

The coefficients were derived from a period when gasoline lead consumption dropped significantly and since then, the trend has continued to an even greater extent. As noted earlier, adults have large stores of skeletal lead accumlated over times of higher lead exposures. Some of this lead is mobile and can be resorbed from bones into the bloodstream after reductions in exposure (Rabinowitz et al., 1977). The extent of such bone lead resorption and the amount of time for re-establishing new steady-state PbB levels in response to declines in gasoline lead emissions since NHANES II is difficult to quantify. The only available direct data on bone lead resorption come from excessively exposed people who may not have the same biokinetic rates as those with lower exposures. Similarly, other studies using isotopic lead tracers may not be relevant since isotopic exchange rates may differ from bone lead resorption rates. Nevertheless, workers exposed for periods up to 10 years (O'Flaherty et al., 1982; Hryhorczuk et al., 1985) and 20 years (Ahlgren et al., 1987) in the lead industry, with PbB levels as high as 70 µg/dl (and normal renal function) had blood lead half-lives between 20 and 700 days and showed significant blood lead reductions as quickly as 3 months to 2 years. Every indication from the NHANES II blood lead/gasoline lead analysis (e.g., short time lags, seasonal parallels), and other studies (e.g., Facchetti et al., 1985) indicate that non-occupationally exposed adult blood lead levels respond quickly (e.g., as quickly as one month) to fluctuations in gasoline lead. Given the fact that by 1990, the current phase of the gasoline phasedown regulation (i.e., 0.10 grams of lead/leaded gallon--current levels are slightly lower; D. Kortum, EPA's Office of Mobile Sources, personal communication) will have been in full effect for about 3 years, whatever changes predicted based on the 1976-1980 NHANES results can reasonably be applied to estimating "baseline" 1990 blood lead levels.

Table C-2 summarizes calculations using the regression coefficients from NHANES II along with gasoline lead to estimate changes in average blood lead expected from changes in recent and future gasoline lead usage. The regression coefficients were similar for the different population groups. As observed in the NHANES II analysis, the continued dramatic decline in

TABLE C-2. PREDICTED CHANGES IN AVERAGE BLOOD LEAD LEVELS ASSOCIATED WITH GASOLINE LEAD TREND

	Children (0.5-5 years)	Women (15-44 years)	White Men (40-59 years)	Black Men (40-59 years)
1978 Geometric Mean PbB (μg/dl)	14.9	10.8	15.4	17.7
Regression Coefficient (log PbB change/100 tons gasoline Pb change)[a]	.1220	.1558	.1216	.1216
Reduction in Gasoline Pb Consumption, 1977-78 to 1990 (tons/day)[b]	435	435	435	435
Predicted Reduction in Gas/Lead Contribution to 1990 Mean PbB (μg/dl)	-8.6	-5.5	-9.1	-10.5

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[a] Calculated by Joel Schwartz.

[b] Estimates listed below, of gasoline lead usage provided by John Holley and Dave Kortum of EPA's Office of Mobile Sources, Field Operations and Support Division; 1990-92 projections based on Turner Mason and Company, 1987 report on petroleum industry marketing:

	1977-78	1988	1990	1992[c]
Tons/day	438	4.8	2.9	1.4

[c] Despite further decline in 1992 in gas-lead consumption, no additional reduction in blood lead levels predicted by model.

gasoline lead emissions is predicted to parallel a major shift in PbB levels. This decline will be used along with adjustments expected because of other lead exposure trends to estimate average 1990 baseline blood lead.

5. Adjustment for Dietary Lead Reductions

A significant reduction in dietary lead intake has occurred since the late 1970's, and this trend will continue as atmospheric lead emissions and deposition, the use of lead-soldered cans, and lead levels in drinking water all continue to decline. Source contributions to children and adult diets are estimated in Chapter 7 of the CD, as described in Section II.D of this paper. Age-specific estimates of dietary lead intake in children for 1978-1985 based on that information are detailed in Sledge (1986) and projections of future lead intakes, incorporating more recent data in Flegel (1988) have been estimated for children in 1990 (Cohen, 1988). Estimated differences in dietary lead intake between 1978 and 1990 are shown in Table C-3. The 1982-85 values are taken directly from Flegel et al. (1988), who showed that earlier predictions using the modeling approach developed in the CD were validated by actual data since made available by FDA. The 1978 estimates are backward extrapolations of the 1982-95 data using the calculations in Sledge (1986). Among children, only 2-year olds are considered for simplicity since other children would have roughly the same proportional decrease in dietary lead over this time period. Likewise, the 1978-1985 extrapolations for adults were calculated by assuming the same proportional declines that occurred in 6-year olds' dietary lead intake, given that the sources of lead intake among older children are the same as for adults (confirmed by Rob Elias of EPA's Environmental Criteria and Assessment Office; personal communication to Jeff Cohen, March 9, 1988). The 1982-1985 estimates for all 3 subgroups are consistent with updated analyses by FDA based on recent Total Diet Study data for different age-sex categories, as presented in the ATSDR (1988) report to Congress (Table IX-6).

TABLE C-3. TRENDS IN DIETARY LEAD INTAKE (μg/day): TOTAL INTAKE (AND ATMOSPHERIC CONTRIBUTIONS)

	1978[a]	1982[d]	1983	1984	1985	1990[e]
2 year old children	52.9 (27.0)[b]	30.6	21.6	21.7	13.1	10.4 (1.2)[f]
Adult Females	65.6 (26.3)[c]	37.9	27.2	27.6	14.5	11.5 (4.3)[c]
Adult Males	91.5 (39.0)[c]	55.2	36.3	42.5	19.8	15.7 (6.1)[c]

[a] 1978 values are extrapolations based on estimates in Sledge (1986), assuming a mean lead in drinking water of 12 μg/L.

[b] Atmospheric contribution to 1978 dietary lead in children from Sledge (1986) based on Multiple Source Food Model developed in CD, Chapter 7.

[c] Atmospheric contributions to 1978 and 1990 adult diets derived by taking proportional contributions to 1982-1985 diets, as provided in Flegel et al. (1988).

[d] 1982-85 values are from Flegel et al. (1988). Exception is 1982 value for children which is derived in Cohen (1988a,b) assuming a mean tap water lead level of 17 μg/L.

[e] 1990 projections, described in Cohen (1988a,b) derived by adjusting 1982 values based on following assumptions: 1) switchover to lead-free cans will continue such that by 1989, lead levels in canned foods will be 10% of 1982 values; 2) average lead level in drinking water will be 17 μg/L in 1990.

[f] Atmospheric contribution to 1990 children's diet derived in Cohen (1988a) by multivariate regression analysis using CD Multiple Source Food Model, FDA data, and projected gasoline and ambient air lead data.

The dietary intake estimates for 1982, based on the CD analysis, were adjusted for the changes that have occurred and that are expected to continue, due to the phaseout of lead-soldered cans, reductions of lead in drinking water, and reductions in atmospheric lead deposition onto crops and other vegetation, to yield 1990 dietary estimates. Again, the same proportional change in dietary lead intake for a 6-year old, estimated between 1982-85 and 1990, can be assumed for adults since the sources of dietary lead in both diets are the same.

Part of the reduction in dietary lead intakes is attributable to reduced deposition of atmospheric lead particles, and although the NHANES II regression coefficients used to adjust PbB levels for the gasoline phasedown represented total exposure, they probably did not capture much of the latter pathway since it takes some time (at least more than a month) for changes in gasoline lead usage to be reflected in different foods. The gasoline lead/blood lead coefficients would likely have been higher had a longer timeframe been analyzed.

Given the uncertainties of precisely how much dietary lead exposure is in fact captured by the gasoline lead/blood lead coefficient, a range of assumptions will be made. As a lower bound, we will assume that the adjustment described previously for gasoline lead reductions, based on 1976 to 1980 data, will account for reductions in atmospheric lead contributions to dietary lead intake. In other words, the entire reduction in dietary intake estimated from 1978 to 1990 (52.9 µg/day - 8.8 µg/day = 44.1 µg/day for children, for example) would be considered in addition to the adjustment for gasoline lead reductions. Alternatively, the contribution of atmospheric lead deposition will be "factored out" of the estimates of dietary lead intake for 1978 and 1990 to account for reductions in gasoline lead usage. Atmospheric contributions to children's dietary lead intake, for example, are estimated to have been 27.0 µg/day in 1978 (Sledge,

1986) and to be 1.2 µg/day in 1990 (Cohen, 1988). Factoring out atmospheric lead deposition from the dietary lead intakes cited above yields 52.9-27.0 = 25.9 µg/day for 1978 and 10.4-1.2 = 9.2 µg/day for 1990. The range of dietary lead intake reductions estimated between 1976 and 1990 for children is therefore comprised of two values--25.9-9.2 = 16.7 µg/day, and 52.9-10.4 = 42.5 µg/day. Corresponding ranges for adult females and adult males are 32.1-55.1 µg/day and 42.9-75.8 µg/day, respectively.

To estimate the impact on blood lead levels that changes in dietary lead have had, and will continue to have by 1990, dose-response relationships between PbB levels and lead levels in food and/or water can be used. Studies relating blood lead to dietary intake, measured by duplicate diets or fecal lead determinations, are summarized in the CD and the most relevant coefficients relating dietary lead, or "slopes" are cited in Section IV.B.1 and IV.B.2 of this report. Most available studies are either on infants or adults; Slopes, in units of µg/dl PbB per µg dietary Pb intake/day, range from about .02 to about 0.060 (U.K. Central Directorate, 1982; Sherlock et al., 1982) from adult studies and for infants, from 0.16 (Ryu et al., 1983) to 0.25 (Lacey et al., 1985) at low levels, and 0.026 at high exposure levels where the relationship flattens out (Marcus, 1989 based on Lacey et al. data).

The Laxen et al. (1987) study on Scottish school children provides data to relate water lead concentration to blood lead. After partialling out the effects of house dust lead, the authors suggested a linear relationship between blood lead and water lead with a slope of 0.062 µg/dl per µg/L. Reanalysis of these data indicated that a piecewise linear relationship fit as well as non-linear models, with a slope of 0.161 µg/dl per µg/L for water lead concentrations below 15 ppb and a lower slope of 0.0318 for water lead above that inflection point (Marcus, 1989). Assuming that the kitchen water lead levels correlated well with total dietary lead concentration, for this population, this relationship can be used to predict the contribution of diet to blood lead. Results of the Laxen et al. analyses can thus be used to predict changes in blood lead by converting dietary lead intake values (in μg/day) to concentrations in terms of μg/kg, or ppb, as follows:

$$\frac{\text{Dietary Lead Intake (μg/day)}}{\text{Total Dietary Intake (kg/day)}} \quad \times \quad \text{slope} \ \frac{(\text{μg/dl})}{(\text{μg/Kg})}$$

where: reduction in dietary lead intake between 1978 and 1990 is in the range of 16.7 to 42.5 μg/day;

total dietary intake.for a 2-year old child is estimated at 1.502 kg/day (CD , Table 7-15); and

slope from the Lacey et al. study is estimated at 0.16 for lead levels below 15 ppb and 0.032 for levels above 15 ppb.

Table C-4 presents results of the above calculations for children, along with adjustments for 1978 adult PbB levels derived from multiplying the slopes discussed in Section IV.B.2 by estimated reductions in dietary lead intakes. These adjustments will be combined with those made previously for the gasoline lead phasedown to yield 1990 average baseline PbB levels.

TABLE C-4. ADJUSTMENTS TO 1978 MEAN BLOOD LEAD LEVELS FOR DIETARY LEAD REDUCTIONS

	Range of Differences Between 1978 and 1990 Estimated Dietary Lead Intake (μg/day)[a]	PbB Reduction Estimated By 1990 Due to Dietary Lead Reduction (-μg/dl)[b]
Children	16.7-42.5	0.9-1.8
Adult Females	32.1-55.1	1.0-1.8
Middle-aged Men	42.9-75.8	1.4-2.4

[a] From Table C-3. Ranges reflect uncertainty as to how much of the adjustment for gasoline lead reductions, using NHANES II-derived coefficients, captures changes in atmospheric contributions to dietary lead intake.

[b] Slope values used to convert dietary lead intake to average blood lead changes are from reanalysis by Marcus, 1989 of Laxen et al. (1987) for children (see text); and 0.032 μg/dl per μg Pb/day for adults (Sherlock et al., 1982; Cools et al., 1976; see Section IV.B.2).

6. Adjustment to Children's PbB for Reductions in Maternal Exposure

Since lead exposure has been, and will continue to be, reduced for women since the 1970's, PbB levels in children born since then can be expected to be lower as a result. Over the course of the NHANES II survey, much of the reduction in gasoline lead emissions was probably reflected in lowered maternal, as well as newborn PbB levels. Thus, it can be assumed that the adjustment to children's PbB already described for gasoline lead changes sufficiently included any gasoline-attributable reductions maternal lead exposure.

Reduction in maternal PbB attributable to changes in PbB dietary lead intake, however, is not reflected in adjustments made previously. One method to account for this is by using a kinetic model fit to blood lead data collected longitudinally from young children from birth to 27 months (Succop et al., 1987). This model provides a rate of blood lead change which can be applied to the amount of newborn blood lead affected by reductions in maternal dietary lead intake estimated between 1978 and 1990. As will be shown in Section C.8 below, the ratio between newborn PbB to maternal PbB is on average around 0.8. Thus, the following equation is derived to account for the propagation of PbB reductions in women, due to dietary changes (1.0 - 1.8 μg/dl), to 2-year olds:

$$[(1.0 - 1.8\ \mu g/dl) \times 0.8] * e^{-\alpha t} = dPbB_m$$

where: the rate constant $\alpha = 0.072$ (Succop et al., 1987)

$t =$ 24 months

$d\ PbB_m =$ PbB reduction in 2-year olds expected in 1990 due to reductions in maternal dietary lead intake.

The resulting range for $dPbB_m$ is 0.2 - 0.3 µg/dl at age 2. This adjustment will be combined with the others for 2 year old children to yield an expected background mean PbB for 1990.

7. Adjustment for Implementation of the Lead NAAQS

The midpoint of the NHANES II survey, 1978, was the year the current lead NAAQS was promulgated. Since then air lead levels have dropped dramatically throughout the country, mainly due to the gasoline phasedown, but also because of implementation of the air standard, especially in areas dominated by industrial point source emissions. Since the bulk of the NHANES II sample lived in urban or rural areas, fairly remote from major lead point sources, it will be assumed that correcting for the gasoline lead phasedown will capture most of the reduction in air lead exposures that has occurred since implementation of the standard.

8. Additive Adjustments to 1978 Blood Lead Level Averages

Table C-5 summarizes the derivation of mean PbB levels estimated for four subpopulations in 1990. These averages reflect changes that have occurred since 1978, and that are expected to continue, in gasoline lead emissions and deposition, canned food technology, and corrosion control for lead in drinking water. Any other changes not quantified were probably of lesser importance for the bulk of the NHANES II population (e.g., reductions in industrial point source emissions). The 1990 baseline averages can subsequently be used to model exposure changes expected under alternative lead NAAQS between 1990 and 1996 using the disaggregate model for adults and the aggregate model for children. The methodologies for these models are discussed in Section V.

Table C-5. DERIVATION OF 1990 "BASELINE" AVERAGE BLOOD LEAD LEVELS

	Children (2 years)	Women (15-45 years)	White Men (40-59 years)	Black Men (40-59 years)
1978 NHANES II Average (µg/dl)[1]	14.9	10.8	15.4	17.7
Adjustment for Gasoline Pb Phasedown (-µg/dl)	8.6	5.5	9.1	10.5
Adjustment for Dietary Lead Reductions (-µg/dl)	0.9 - 1.8	1.0 - 1.8	1.4 - 2.4	1.4 - 2.4
Adjustment for Reductions in Maternal Exposures (- µg/dl)	0.2 - 0.3	-	-	-
Estimated 1990 Average (µg/dl)[1]	4.2 - 5.2	3.5 - 4.3	3.9 - 4.9	4.8 - 5.8

[1] Geometric Mean Levels

9. Estimating Fetal Exposures

Different indices of prenatal and neonatal lead exposure have been found in longitudinal studies to correlate with delays in early mental and physical development. Lead readily crosses the placenta and accumulates in fetal tissues throughout development. The dynamics of lead exchange during pregnancy are complex and poorly understood (see CD Addendum). Ideally, serial measurements of maternal lead would have been taken in the studies just prior to conception and throughout pregnancy and biokinetic models would exist that can estimate actual fetal exposures. In the absence of either, the question is whether equilibrated PbB levels in pregnant women are adequate surrogates for critical fetal lead exposure, or is there a better index.

Umbilical cord blood lead, for example, which has been a key effects predictor in several studies, could be estimated by adjusting maternal PbB predictions and applied to available dose-response relationships to assess risks. Table C-6 lists available data on paired mother/child PbB concentrations along with ratios between average maternal and cord PbB levels. The consistently high ratios (mean of 0.80 or 0.82, depending on the inclusion of one study with outlying results) indicates that the mature fetus absorbs a sizable fraction of circulating maternal lead.

Cord blood lead, however, may not accurately reflect individual circumstances or past exposure levels. In at least some pregnancies, there could be greater than normal transfer of lead from mother to the fetus. Mobilization of bone lead stores during pregnancy and lactation may be more substantial in some women, and iron and calcium deficiency, both common in pregnancy, enhances gastro-intestinal absorption of lead (Rom, 1976). In addition, integrated exposure levels during the course of pregnancy

Table C-6. MATERNAL: CHILD BLOOD LEAD CONCENTRATIONS AND RATIOS

Study/Location	Number of Subjects	Mean Blood Lead (μg/dl) Maternal	Cord	Newborn	Ratio of Cord: Maternal PbB
Barltrop (1968)/U.K.	29	13.9	10.8	-	0.78
Harris and Holley (1972)/U.S.	24	13.2	12.3	-	0.93
Haas (1972)/Germany	294	16.9	14.9	-	0.88
Zetterlund et al. (1977)/4 areas in Sweden					
A	21	6.1	4.4	-	0.72
B	173	9.2	8.0	-	0.87
C	103	8.4	7.3	-	0.87
D	37	9.4	6.8	-	0.72
Clark (1977)/Zambia					
- Control	31	14.7	11.8	-	0.80
- Mine	122	41.2	37.0	-	0.90
Hubermont et al. (1978)/ Belgium	70	12.0	10.1	-	0.84
Buchet et al. (1978)/ Belgium	474	10.4	8.3	-	0.80
Dragovic (1980)/ Yugoslavia					
-Smelter	102	32.6	28.3		0.87
-Control	45	18.6	17.3		0.93
Alexander and Delves (1981)/U.K.	165	12.2	-	11.0	-
Moore et al. (1982)/ U.K.	236	14.0	12.0	-	0.86
Winneke et al. (1985)/ FRG	114	9.3	8.2	-	0.88
Ernhart et al. (1986)/ U.S.	118	6.5	5.9	10.0	0.91
McMichael et al. (1986)/ Australia					
Port Porie	519	10.8	10.1	11.2	0.94
Other	172	7.7	6.2	7.5	0.81

Table C-6. MATERNAL: CHILD BLOOD LEAD CONCENTRATIONS AND RATIOS (Continued)

Roels and 1978 Lauwerys/Belgium	474	10.1	8.4	-	0.83
Dietrich et al. (1987)/ U.S.	305	8.0	6.3	4.6-5.9	0.79
Bellinger et al. (1986)/ U.S.	216	-	6.5	6.2-7.7	-
Milman et al. (1988)/ Denmark	48	35	20	-	0.57
				Weighted Mean:	0.82
Fahim et al. (1976)/ U.S.					
Region I	240	13.1	4.3	-	0.33
Region II	177	14.3	4.6	-	0.32
				Weighted Mean:	0.80

may not be accurately indexed by blood lead levels at delivery or by single measurements during pregnancy. Average maternal PbB levels can fluctuate considerably during pregnancy (CD Addendum, p. A-45), while the equilibrium between blood and soft and skeletal tissues in the mother is displaced, and lead is transferred to the placenta and fetal tissues; Alexander and Delves (1981) found an almost 20% drop in maternal PbB levels from 8 weeks until delivery.

As noted in Section V.A., there are significant uncertainties associated with predicting PbB levels among pregnant women and certainly fetal lead exposures, under alternative regulatory scenarios. Further, the chance of underpredicting exposures is great. Therefore, for purposes of assessing risks associated with alternative lead NAAQS, no quantitative estimates will be generated of PbB levels among pregnant mothers as surrogates for fetal exposures. Potential risks to this extremely sensitive population will be emphasized in qualitative terms.

APPENDIX D. CASAC CLOSURE REPORT

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

April 27, 1989

OFFICE OF
THE ADMINISTRATOR

The Honorable William Reilly
Administrator
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

Dear Mr. Reilly:

We are pleased to transmit via this letter the advice of the Clean Air Scientific Advisory Committee (CASAC) concerning its review of the EPA document "Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation" (August 1988).

This document was reviewed by the Lead Exposure Subcommittee of CASAC on October 25, 1988. It was the unanimous consensus of the Subcommittee that the document is scientifically adequate for use in the standard setting process for lead as an ambient air pollutant. The CASAC hereby endorses the report of its Subcommittee. A detailed presentation of our views are contained in the attached report.

We appreciate the opportunity to provide advice on this important issue. Further advice concerning the lead national ambient air quality standards will be contained in our closure letter on the Lead Staff Paper.

Sincerely,

Timothy Larson

Timothy Larson
Chairman, Lead Exposure
Subcommittee

Roger O. McClellan

Roger O. McClellan
Chairman, Clean Air Scientific
Advisory Committee

U. S. Environmental Protection Agency
Science Advisory Board
Clean Air Scientific Advisory Committee

LEAD EXPOSURE SUBCOMMITTEE

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REPORT OF THE CLEAN AIR SCIENTIFIC ADVISORY COMMITTEE ON ITS REVIEW OF "REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR LEAD: EXPOSURE ANALYSIS METHODOLOGY AND VALIDATION"

With the dramatic decline in the emissions of lead from mobile sources, there has been increased interest in airborne lead near stationary sources. Given a proposed change in lead concentrations near stationary sources due to reduced emissions, will there be predictable changes in blood lead levels in the surrounding population? Exposure models are currently the only practical tool that can address this question within the framework of the national ambient air quality standard (NAAQS) setting process. The scientific framework for such modeling is the basis of the document reviewed by the Clean Air Scientific Advisory Committee's (CASAC) Lead Exposure Subcommittee. In addition to discussing various modeling approaches, the document also presents several validation studies in order to compare these modeling approaches with actual observations of blood lead levels near several point sources of lead. It was the unanimous consensus of this Subcommittee that the document is scientifically adequate and that the EPA staff's proposed changes to the document discussed at the meeting appropriately address the written comments of the Subcommittee members. The Clean Air Scientific Advisory Committee (CASAC) hereby endorses this report of its Lead Exposure Subcommittee.

The validation studies presented convincing evidence for a decrease in blood lead levels with increasing distance (out to several kilometers) from a point source. The Subcommittee agreed with the conclusion that any attempt to predict blood lead levels must include all the important exposure pathways and that the direct inhalation route of airborne lead is a relatively minor pathway in children. The validation studies also provided additional information on the lead levels of other important exposure media, including soil, house dust, food and water. Therefore, these studies provide a unique opportunity to test the ability of various exposure/uptake models to predict mean values of blood lead from various routes of exposure. Blood lead levels were predicted using both a disaggregate approach as well as a biokinetic approach. The Subcommittee recognized that several of the inputs to the exposure models are uncertain, but felt that this uncertainty was adequately recognized in the document. More important, the Subcommittee concurred with the general modeling framework and endorsed the use of the biokinetic model in children under six years of age and the use of the disaggregate approach in adults. The Subcommittee also strongly emphasized that these modeling predictions were not valid for pregnant women and their fetuses due to a lack of information on this potentially important subpopulation. The Subcommittee also recommended that the exposure model outputs include not only the predicted mean blood lead levels as a function of downwind distance but also the corresponding lead levels in all exposure media including air, soil, dust, food and water. These outputs would provide an additional basis for evaluating model performance.

Given a predicted mean blood lead level, another important component of the exposure model is the prediction of maximum blood lead levels in exposed individuals. EPA's approach is to use empirical estimates of the variance of blood lead levels in the general population, as well as those in populations living near lead point sources, as a predictor of peak to mean values. The document correctly recognized that the population variance estimates depend on many different factors (e.g., biological, climatic, behavioral) that exposure modeling cannot fully capture at this time. Given the uncertainties, the Subcommittee agreed that the only reasonable assumption is to use the range of variance estimates from the empirical data. However, because this is a sensitive parameter, we felt the additional concern that as blood lead levels continue to decrease in the future, the assumption of a constant proportional variance (log-normality assumption) may be compromised by analytical uncertainties in the measurement of blood lead, but the Subcommittee felt that this issue was adequately addressed in the document.

The Subcommittee felt very strongly that the results of this modeling exercise not be taken out of context. For example, because the available data on lead in drinking water for the validation studies was limited, the biokinetic model in this application was used to calculate average drinking water exposures over time. However, the biokinetic model is sensitive to total intake from this route and can account for variations in water lead exposure where appropriate data are available. While the model can be used now to evaluate relative changes in blood lead levels from changes in water lead levels, it has not been calibrated for absolute assessments of risk from drinking water in the same way as done for other routes of exposure. Use of the model for other metals was also not recommended at this time. In addition, although the Subcommittee agreed that an appropriate application of this approach might be for prediction of offsite lead exposures from fugitive dust emissions, there was concern that until non-linearities in the relationship between lead exposure and blood lead are incorporated into the model, the model be limited to use in areas where soil lead levels do not exceed 4000 ppm. In addition, the model should not be used in areas where ingestion (pica) of paint fragments is an important route of intake because this variable was not considered in the case study validation. Finally, the biokinetic model should not be used for predicting adult blood lead levels at this time due to limited data regarding historical exposures and the possible confounding factor of blood lead coming from bone.

The Subcommittee was also asked for guidance on several technical issues that are summarized below. As to the range of dirt ingestion rates used in the report (55-135 mg/day depending on age), the Subcommittee agreed that this is a relatively poorly defined parameter subject to climatic variations. Some members felt that the value of 100 mg/day represented an upper limit for a high risk child, whereas others felt that the use of the Binder et al. and Clausing et al. studies was as good a choice as any until further data are available. All members agreed that this is an important parameter in determining total intake and that the uncertainties have been adequately discussed in the document. In this regard, there was agreement that the emphasis in future research should focus on the lead levels in the surface layer of the soil and not on the older, deeper layers. There was general agreement that the model use a constant soil lead level in predicting future scenarios, but that the house dust component should track the air lead value. Finally, the approach of interfacing the biokinetic and disaggregate models for intermediate age groups was judged acceptable by the Subcommittee in the absence of any other available information to the contrary.

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